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MESSAGE

It is with immense pleasure that I have learned that Dr.G.M.Malik is bringing out a Medical Journal first of its kind and I must congratulate Dr.Malik for his endeavour to bring out his medical Journal in the State. This action of his will be remembered for long time to come. He will be remembered as pioneer in the Medical History of this State. I am sure the contributors will contribute articles of high calibre thereby keeping medical profession well informed about the latest developments in the field of Medical profession and allied technologies.

I wish Dr.Malik success in this endeavour.

(G.Q.Allaqaband)

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Editor's View

This is the first issue of JK-PRACTITIONER. This Journal will continue to provide Medical Education in a concise, authoritative, as well as up-to-date reviews of every day problems in Medicine, surgery & allied subjects for the need of students, general practitioners, specialists & teachers.

In every issue you will find interesting articles, case reports, diagnostic tools, meet the professor, scanning of latest journals, books as well as letters to the editor. Very soon stellite type of consultation (replies to questions related to your day to day practice) will be started. I am greatly thankful to all my colleagues in the country as well as abroad for helping me to fulfill my dream to start this journal from foot hills of Himalyias & bring it at international level in near future. Last but not the least, please send your suggestions, opinions, contribution for the future issues.

Dr.G.M.Malik
M.D.
Chief Editor.

HEPATITIS-E

"the enterically transmitted non-A, non-B hepatitis"

Professor M.S.Khuroo

Hepatitis E is an ecologically determined, self limiting enterically transmitted, acute viral hepatitis that occurs most frequently in epidemic outbreaks in developing countries. Hepatitis E also accounts for a significant number of sporadic cases in endemic regions. The disease occurs in adults between 15-45 years of age with lesser attack rate in children and elderly. Hepatitis E has increased incidence and mortality in pregnant women. Hepatitis E is etiologically associated with newly identified and molecularly characterized agent, the hepatitis E virus (HEV)[1-2].

Historical Background

Hepatitis E was first recognized during an epidemic of hepatitis which occurred in Kashmir (India) in 1978[1]. Th epidemic involved an estimated 52,000 cases of icteric hepatitis with 1650 cases of fulminant hepatic failure and 1560 deaths. In 183, Mickail Balayan of the Institute of Poliomyelitis and Viral Encephalitis Russia, reported on the self ingestion of acute phase stool suspensions of patients from a waterborne epidemic of non-a hepatitis in Central Asia[3]. He described the resultant clinical hepatitis, recovery of 27-32nm virus like particles (VLPs) from his feces and development of antibodies to these particles during convalescence. Furthermore, he transmitted the virus to cynomolgus monkeys thereby establishing an animal model[5]. Since then, the viral genome has been cloned, sequenced and an enzyme linked immunosorbent assay for the detection of antibodies to HEV has been developed for the diagnosis and seroprevalence of hepatitis E (Table-1).[4,5,6]

Hepatitis E Virus

HEV has a particle size of 32-34nm with spikes and indentations on its surface (Table-2). The genome of the virus is a single positive stranded, polyadenylated RNA of approximately 7.5 Kb. Three open reading frames (ORFs) have been postulated so far. The first ORF (ORF1), begins 28 nucleotides from the 5' end and extends 5079 bp before ending at nucleotide position 5017. The ORF2 begins at nucleotide position 5147 and extends 1980 bp before terminating 65 bp upstream of the poly (A) tail. ORF3 encompasses 369 bp, overlaps the first reading frame at it's 5' end by one nucleotide and significantly overlaps ORF2. ORF1 codes for the non-structural region of the virus, ORF2 codes for the structural region of the virus while's the function of ORF3 is not known. The biophysical properties of the virus are consistent with the notion that HEV either belongs to the family Caliciviridae or an entirely new class of viruses (Table-3)[4]. The nucleotide sequence the whole HEV genome has been identified for HEV strains from Burma, Pakistan and China which showed a very less degree of divergence[5,7,8].

HEV has been transmitted to two human volunteers, a number of primates and other animals namely African green monkey (Cercopithecus aethiops), tamarins (Saguinus mystax), cynomolgus macagues (Macaca fascicularis), rhesus monkey (Macaca mulata), Owl monkey (Aotus trivirgatus), Chimpanzees (Pan troglodytes) and marmosets [7,9,10,3,11,12,13,14,15,16,17]. Transmission in Piglets and lamb has also been reported recently. Serial passages of the disease has also been performed in cyno monkeys resulting in similar biochemical and histological findings[7]. The fecal extracts of patients collected during the early acute phase of the disease are used as inoculum and transmit the disease through both enteric as well as parenteral route.

Monkeys develop transient biochemical (transaminitis) and histological (necro-inflammation) hepatitis in peaks between 2-9 weeks post inoculation. Spherical 27-30nm VLPs visualized by Immune-electron Microscopy (IEM) are excreted in stool during preclinical and early clinical phases of the disease. The VLPs in the stool are very few in number and often in degraded form. However, HEV particles have been found in large number in gall bladder bile ten days post-inoculation and continue to be present upto 2 days after normalization of serum transaminase levels. Polymerase Chain Reaction has been used to demonstrate HEV RNA in the blood during late incubation period and within the first ten days of the clinical disease[10].

HEV infection is associated with IgG and IgM antibodies to HEV. IgM antibodies come-up early in the disease and last for a period of 3-6 months. However, IgG anti-HEV seroconversion occurs during the clinical disease and is long lasting. There is evidence that IgG anti-HEV antibodies are protective in nature[19].

Epidemiology

Hepatitis E exists as a sporadic disease with periodic occurrence of epidemics. Epidemics of hepatitis E are of frequent occurrence in developing countries especially in the Indian subcontinent (Table-4). Hepatitis E in these countries is a major national health problem as these epidemics involve tens of thousands of cases[2,20,21,22]. These countries share poor socioeconomic and hygienic conditions and provoke epidemics by contamination of water supplies. The epidemics occur in the rainy seasons of after floods.

The epidemic hepatitis E occurs in unimodal outbreaks with a highly compressed curve of incidence or more prolonged epidemics with multiple peaks of incidence 1,21,23. The secondary attack rate among exposed household members is low. Highest attack rate among cases with clinically overt disease is observed between 15-40 years of age. The most conspicuous feature of epidemic hepatitis E is a very high incidence and severity of the disease among pregnant women[24].

Non-A, non-B, hepatitis is the commonest cause of acute sporadic hepatitis in developing countries. 53% of acute viral hepatitis, 87% of subacute hepatic failure (SAHF) and 60% of cases with fulminant hepatic failure (FHF) are unrelated to hepatitis viruses A, B and C[22,25]. Recent serological data have shown that hepatitis E constitutes over 60% of cases of acute viral hepatitis/ fulminant hepatic failure of non-A, non-B and non-C types in these countries[26].

Some sero-prevalence studies have shown a higher rate of anti-HEV in the general population[27,28]. Sero-prevalence study conducted in Nepal revealed a prevalence rate of 13.9% in general population with 9.2% in the first decade, 13.1% in the second decade, 12.4% in the 3rd decade and maximum prevalence of 27.3% in the 4th decade.

The sero-prevalence survey of hepatitis E in Hongkong showed a prevalence of 16.1% in the healthy subjects[27], while the prevalence of anti-HEV in Turkey has been reported as 5.9%[29]. Hepatitis E was also shown to be responsible for 6.8% of cases of acute viral hepatitis in Spain[30]. These results suggested that HEV is present in Spain and accounts for a low proportion of acute non-A, non-B, non-C hepatitis. A seroprevalence study of hepatitis E in general population was conducted in Kashmir Valley. (Table-5).

Overall prevalence of anti-HEV was found to be 4% only. Antibodies were not detected in any of the subjects in the first two decades and reached a maximal prevalence of 8% in the fourth and fifth decades. In contrast, the overall prevalence of anti-HEV was 89.6%. Anti-HEV Antibodies were detected in 70% of the subjects in the first decade and in over 90% from the second decade onwards.

Disease Pattern

The clinical disease in humans in typical of viral hepatitis, however, cholestasis is a predominant feature in 20% patients and should be differentiated from large bile duct obstruction[1]. The incubation period of hepatitis E varies from 15-50 days and in the case of a human volunteer clinical symptoms developed 36 days after oral intake of infected fecal samples[9]. About half of the patients develop fever and two-thirds complain of arthalgia. Liver function tests are indicative of hepatic necrosis. The billirubin and transaminase rise is comparable to that in acute hepatitis B. Transaminase elevation is monophasic. The disease is self-limiting with eventual clinical recovery is non-fetal cases.

Hepatitis E and Pregnancy

Icteric hepatitis E characteristically occurs more commonly and with great severity in pregnant women. In fact, the major cause of mortality in the epidemic disease is the high rate of fulminant hepatitis in pregnant women. Increased serverity of viral hepatitis in pregnant women has been reported from the Indian subcontinent, IRan and many other developing countries[31,32,33]. These data were looked as with skepticism as selection bias could not be ruled out in the hospital data which formed the basis of these reports. Data collected from an outbreak of hepatitis E in Kashmir (1981) showed that the frequency of hepatitis was greater in pregnant (17.3%) than in non-pregnant (2.1%) women and in men (2.8%). The frequency of the disease in the first, second and third trimesters was 8.8%, 19.4% and 18.6% respectively. Severe disease in the form of fulminant hepatic failure developed more often in pregnant women (22.2%) than in non-pregnant women, none of whom had this complication, and in men(2.8%). Fulminant hepatitis developed exclusively in the third trimester. The nutritional status of women in the area affected by outbreak or those women who developed fulminant hepatic failure was assessed as good. The overall mortality in pregnant women was found to be 20%. The reason for this high mortality is not clarified[24].

Hepatitis E, A Disease of Adult Population

Hepatitis E in epidemic and sporadic form has certain features in common. There is high attack rate in adults (15-40 years) with lower attack rate (1.2%) in children below 14 years. These data are in contrast with hepatitis. A disease which in the developing countries occurs preferentially in the childhood. The occurrence of hepatitis E in children has been focuses in one study from Egypt[34]. Hepatitis E virus was the etiological agent in 8/36 Egyptian children, majority (77%) of the children with acute non-A, non-B, non-C were unrelated with hepatitis E. In another study from Hongkong only one of 81 patients with acute viral hepatitis in the age group 11-20 years had positive IgM anti-Hev against 7% and 10% positively in the age group of 21-30 and 31-40 years respectively[27]. These data point to the fact that although children are not immune to hepatitis E its occurrence in this age group is scare/rare. It has been postulated that hepatitis E in children manifests as anicteric disease and is not apparent clinically. However, recent data collected during an epidemic of hepatitis E in Pinglina (Kashmir) showed that the prevalence of inapparent infection in children was 7.3%) against 22.2% in adults). The total attack rate (sum of icteric and anicteric cases) in children was 7.3% verses 24.0% in adults. This disproved the hypothesis that children suffer from inapparent hepatitis E infection (unpublished data - personal observations).

Mode of Transmission

There is convincing epidemiological evidence that hepatitis E is a water-borne disease[1]. The epidemic curve is unimodal and highly compressed, indicating a single common source of infection. Water supply used by the affected population during an epidemic is usually grossly contaminated and epidemics occur in seasons when water contamination is expected to be maximal e.g. during winter months in Kashmir when the water level falls, reducing dilution of contaminants and following heavy rains during the monsoon season in the plains of India and Nepal when there is fecal contamination in the plains of India and Nepal when there is fecal contamination of water sources[2].

It is important from the public health point to know the mechanism of water contamination so that adequate protective measures may be instituted. Contamination of water has been documented in many environmental settings e.g. (i) heavy rains, (ii) flooding and recession of flood water, after which rivers change course, (iii) contamination of drinking water sources (rivers, streams, unprotected wells) with raw sewage and contaminated surface waters from open drains, (iv) leaking water pipes with intermittent flow running through polluted soil or interconnected with raw sewage disposal pipes and (v) crowded living conditions with unsafe water supply and disposal of human wastes as in refugee camps and rapidly growing urban slums[2].

Following epidemics, secondary waves of hepatitis do not occur, suggesting that person to person contact transmission is not a major factor in the evolution of the outbreaks. However, person to person spread presumably does occur in some epidemics on NANGH which are of protracted type, since new cases of jaundice appear for months after the main epidemic is over, moreover, sporadic form of NANGH is also common[2].

Although hepatitis E is predominantly an enterically transmitted disease, parenteral route of transmission may occur under special circumstances[9,35]. Of the 53 patients of hepatitis E studied, the disease in 3 patients was possibly parenterally transmitted though transfusion[26]. PCR performed for HEV RNA established that viremia occurs during preicteric phase and is detected upon about ten days after the onset of clinical disease. The presence of HEV in blood suggests that possibility of sporadic transmission of hepatitis E in endemic areas via parenteral route. Hepatitis E has been transmitted in Rhesus monkeys by pooled acute phase sera from enterically transmitted NANBH patients and one hospital outbreak of hepatitis E also points to parenteral route of transmission[12,35].

Liver Histology

Liver biopsies in patients with hepatitis E reveal portal triaditis, cholestasis, lobular inflammation and degeneration of varying degrees, suggestive of acute viral hepatitis. However, nearly half of these patients have distinctive morphological changes designated as Cholestatic Viral Hepatitis[1]. The changes include: absence of diffuse lobular degeneration and inflammation; predominance of focal necrosis; marked intrahepatocytic and intracanalicular cholestasis with characteristic bile plugs in the lobules, leading to pseudo-ductular formation around these; and portal triaditis with mono-nuclear cells infiltration with occasional polymorphonuclear leukocytes. These histological features in acute viral hepatitis were reported from the West[36,37] and by Gupta and Smetna in the 1955-56 Delhi epidemic[38]. Patients with cholestatic hepatitis cannot be differentiated from the usual histological type by age, sex, clinical picture, occurrence of disease in pregnancy, serum bilirubin and transaminase levels etc. The occurrence of both histological types in the same outbreaks excludes the possibility of two viruses, each causing different types of liver injury[1,2]. Liver histology in experimentally infected animals showed acidophilic types of hepatocytic degeneration and focal hepatic necrosis accompained by accumulation of mono-nuclear macrophages and activated Kupffer cells, a less striking feature was the presence of lymphocytes in inflammatory infiltrates. Focal necrosis and intralobular and portal inflammatory infiltrates have also been observed in other species of HEV-infected primates (rhesus monkeys, tamarins, chimpanzees). The cholestatic morphological changes observed in patients did not develop in macaques, chimpanzees, or tamarins with elevated aminotransferase activity. The hepatocytes surrounding focal necrosis underwent injuries with the disruption of cytoplasmic organelles under the electron microscope. Such hepatocytes are in contact with lymphocytes and have virus particles. The majority of these lymphocytes are positive for cytotoxic/suppresser phenotypes by immuno-staining[39].

Electron microscopy of liver cells from infected marmosets showed cisternal dilation of the endoplasmic reticulum, irregularly-shaped nucleus and disorganization of the mitochondrial

HEV outbreak in Somalia, 125 specimens were found reactive specimens (122 were also found positive with synthetic peptides) indicating a concordance of approximately 98%[40]. In the Kashmir outbreak of 1978-79 IgG anti-Hev was detected in 71% of 45 patients and 75% of these reactive specimens also showed IgM anti-HEV[41].

The diagnosis of hepatitis E infection is established by the presence of IgM antibody to HEV in a single serum sample. Diagnosis of hepatitis E can also be made by seroconversion of IgG anti-HEV in the convalescent sera. In some patients of hepatitis E infection IgG anti-HEV may be detected in the acute phase sera and disappear in the convalescent sera.

Samples collected very early from a small number of acute viral hepatitis patients (NABCD) do not show either IgM or IgG anti-HEV. But samples collected late from the same patients either during late acute phase or early convalescent phase show the presence of IgG anti-HEV. The seroconversion in these patients also indicates a Hev infection.

Presence of IgG anti-HEV alone in a specimen from an acute viral hepatitis patient can be taken as a presumptive marker for hepatitis E. This is so because the overall seroprevalence of anti-HEV in the general population has been found very low.

The seroprevalence data can be obtained by testing general population for IgG anti-HEV antibodies.

Control and Immunoprophylaxis

As hepatitis E is an ecologically determined disease, its control would depend upon improved sanitation, proper sewage disposal and sup0ply of safe potable water. Mass education during and between outbreaks advising the public to follow proper hygienic precautions and to use boiled drinking water may be helpful. As the morbidity and mortality is seen mainly in pregnant women, special attention is needed in this population during epidemics. Use of immune-serum globulim considerably reduced mortality, when used in 3rd cristae. Also noted was interaction between lymphocytes and hepatocytes where these two types of cells were in close contact without intervening endothelial cells. The microvilli of the hepatocytes were degenerated and lost. the microvilli of the bile canalicli were normal. No virus particles were detected in the liver specimens of marmosets by electron microscopy[15].

Diagnosis

The diagnosis of hepatitis E uptill recently was done by exclusion of hepatitis A, B and C and epidemilogical feature. However, the methods used for diagnosis of hepatitis E are listed in Table-6.

ELISA for detection of IgM and IgG anti-HEV is the most convenient, inexpensive and a suitable assay for routine diagnosis of hepatitis E as well as for sero-epidemiological surveys.

The ELISA has been developed using clonal recombinant HEV antigens based on the sequence of Burma and Mexico strains[6]. Various regions of the hepatitis E virus genome have been cloned and expressed in E.coli and fusion proteins with glutathione Stransferase. Four recombinant antigens, two derived from a Burmese strain of HEV and two derived from a Mexican strains have been shown to contain antigenic sites recognized by individuals with previous exposure to HEV[34,40].

In addition to the recombinant antigens, synthetic peptides based on the sequence of various regions of the viral genome are also used as antigens in the ELISA system. A lot of data has been generated which supports and validates the use of ELISA's using recombinant antigens and synthetic peptides for detection of anti-bodies of HEV. The specificity of the ELISA's using recombinant antigens was demonstrated by the observation the 378 of 386 (98%) specimens obtained from a region considered as non-endemic for HEV infection were negative for antibodies to HEV while as out of 151 specimens obtained from trimester pregnant women in one outbreak in Kashmir[2]. In a controlled study, single dose of immune serum globulin did not significantly reduce the incidence of anicteric hepatitis in subjects receiving the drug when compared to the control group[42]. Recently we used low dose immune serum globulin in contacts of patients with hepatitis E to prevent the possible contact associated transmission. Low dose immune serum globulin was unable to block the contact associated transmission[43].

Vaccination is important for endemic areas/developing countries where hepatitis E is endemic and for tourists who visit these countries. Unless virus neutralizing antibody is elicited by synthetic or recombinant peptides, the attenuated HEV must be utilised for active vaccination. Manipulation of cDNA is absolutely necessary for production of the attenuated HEV for vaccination[44].

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Table-1

Hepatitis E: Historical background

1980	*Description of epidemic non-A, non-B hepatitis (Khuroo MS)*
1981	*Association of fulminant hepatitis in pregnant women with HE infection (Khuroo MS).*
1983	*TRansmission studies and visualization of virus like particles (Balayan et al).*
1990	*Molecular cloning and sequencing of the viral genome (Reyes et al).*
1992	*ELISA for anti-HEV (Yarbough, et al).*

Table 2: Physico-chemical properties of HEV

Morphology:	*Spnerical, non-enveloped particle, spikes and indentations on particle surface, disintegrated by CaCl, freeze-thawing and pelleting.*
Size:	*32 to 34nm*
Genome:	*Single positive stranded RNA, 7.6 Kb approximate. Polyadenylated with three ORFs (partially overlapping): molecular structure -5'-NS-S-Poly A-3'*
Sedimentation coefficient:	*183 S*
Buoyant density:	*1.29gm/cm3 in K Tar/Gly gradient*
In vitro cultivation:	*Several FRhK-4 continuous cell lines established for HEV cultivation - co-cultivation with primary kidney cells derived from HEV-infected primates.*
in vivo:	*No effect on suckling mice inoculated intracerebrally-(10% wt/vol stool suspension).*

Table-3: Classification of HEV

	Genome Organization	**Envelope**	**Sub-genomic RNA(s)**	**Poly-A**
Hepatitis E virus	5'-NS-S-3'	No	Two	Yes
Caliciviridae	5'-NS-S-3'	No	Yes	Yes
Picornaviridae	5'-S-NS-3'	No	No	Yes
Togaviridae	5'-NS-S-3'	Yes	Yes	Yes
Flaviviridae	5'-S-NS-3'	Yes	No	No
Coronaviridae	5'-NS-S-3'	Yes	Multiple	Yes

Table-4:

Site	**Year**	**Icteric cases**	**Attack rate(%)**	**Mortality Overall**	**(%) Pregnant**
Kashmir	1978-82	52000	1.65	3.0	22.2
Delhi	1955-56	29300	2.2	-	10.1
Kanpur	1991	79000	3.7	-	-
Nepal	1973-74	10000	-	-	-
Somalia	-	2000	8.0	4.3	22.2
Burma	1976-77	20000	-	-	18.0
Kirgil (Kazakistan)	1955-56	10000	-	-	18.0
Mexico					
Algeria	1980-81	788	1.1	1.1	-
Pakistan	1987		13.3	20.0	-

Table-5: Sero-prevalence of anti-HEV in 250 healthy subjects in Kashmir.

Age group	No. tested	IgG anti-HAV No (%)	IgG anti-HEV No (%)
0-10	50	35 (70)	0 (0)
11-20	50	46 (92)	0 (0)
21-30	50	48 (96)	2 (4)
31-40	50	47 (94)	4 (8)
>40	50	48 (96)	4 (8)
Total:	250	224 (89.6)	10 (4)

Table-6: Methods used for diagnosis of Hepatitis E

Diagnostic Assay	Basis	Comments
Immune Electron microscopy	1. Detects 27-34nm VLPS In stool specimens antibody.	- Acute phase sera are reactive than convalescent sera.
	2. Detects anti body titers in serum.	- Not a satisfactory test for for routine diagnosis.
		- Expensive equipment required. - VLPS infrequent in stools.
Immune-fluorescence Method.	1. Can determine antigen in liver tissue	- Cumbersome and not suitable for routine purposes.
	2. Can determine antibody in the sera.	
Polymerase chain reaction (PCR)	Detects the viral genome in feces, serum, bile and liver tissue.	Primarily a research tool and cannot be used as routine diagnostic test. Very expensive.
Enzyme linked-Immunosorbent assay	Detects IgM and IgG antibodies to HEV	- A very convenient and simple method for diagnosis and sero-prevalence studies.

TYPHOID IN 90'S

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Introduction

Typhoid fever is widely prevalent in under developed countries of tropics and south east Asian region[1-2]. The disease has been almost eradicated from West to a large extent yet, because of factors like poor hygiene, mal-nutrition low socio-economic status, and inadequate sanitation it still remains a mojor health problem in under developed countries with associated high morbidity and mortality. Ever since the first epidemic of its kind from Asia[3] more and more studies of chloramphenicol resistant typhoid fever and acquisition of multi drug resistance by Salmonella typhi have been published from various places from India and abroad[4-6].

Although chloramophenicol resistance has been reported as early as 1950[6] many studies have been published from time to time in this regard from all over the globe. Presently multidrug resistant enteric fever has taken an Endemic form in India as per published data.

From different studies the incidence of drug resistant enteric fever varies from 0.5% in Netherland[7] to 43% in Los Angles[8] and 49% in, South India[9]. However, our experience during last 5 years we have observed 100% resistance to conventional drugs.

Simultaneously also there have been reports of changing patterns of presentation of this diseases itself making it more important for clinicians to be updated with the newer and diversified aspects of typhoid fever. Since typhoid is known is several odd forms no distinctive clinical signs or symptoms are usually present and the initial clinical diagnosis will largely dependent a high index of suspicion. Any patient with prolonged fever bizzare symptoms, confusional state or delerium should alert the physician to suspect typhoid fever.

Gastrointestinal symptoms : like diarrhoea and vomiting mimic acute gastro enteritis. Patient of typhoid with predominant chest sign ar often diagnosed as acute bronchitis, while patient with predominant neuropsychiatric manifestation and commonly labelled as case of encephititis, or occasionally were treated as psychosis.

With the introduction of newer quinolones and availability of advanced generation cephalosporins the choice of effective treatment of typhoid fever has also enlarged. However, resistance to these drugs has also been noticed though presently at a very low but alarming rate, it may not be difficult to anticipate quinolones and cephalosporins being listed in drugs to which Salmonella is resistant.

Disease Profile

With hundred of patients of suspected typhoid fever getting admitted to our hospital in last 6 years, typhoid fever occurred more frequently in younger people with male predonderance.

Diagnosis was made from history of fever usually of prolonged duration invariably with speenomegaly, & a positive blood cultures for salmonella typhoid and/or favourable serological test. The usual step ladder pattern of fever was not seen, but patients invariably start with high grade fever persisting for many days and at times for many weeks defying the classical self limited pattern of disease. Spleenomegaly appeared not infreq unently appeared in the first week of illness. Toxemia was more pronounced and prolonged. Relative bradycardia was not seen in approximately half of the patients while various G.I.Symptoms-constipation, diarrhoea, abdominal pain, vomiting was more frequent.

Blood culture remained positive for much

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longer period and so were the results of cultures of stool and urine. Many patients had taken different conventional antibiotics in adequate doses and for a reasonable period without any improvement. Chlora-mphenicol, Ampicillin, Furazolidine Cotrimoxozole, even Ist generation Cephalosporivs had all been tried alone or in different combinations.

Blood cultures revealed salmonella typhiod which was identified as phage type, Botype II untypable (USV[2]), and disc sensitivity revealed resistance to chlorampheicol ampicillin, cotrimoxozole tetracycline, and sensitivity to quenolones, frequently to gentamicin and cephalosporins. Rarely disc sensitivity revealed resistance to quinolone as well.

Complications

Clinical course tended to be protracted with high complication rate. (Table I). Some developed these complications very early in the course of disease and presented to hospital with such complications, while many developed these complications latter in the course of illness and few even after completion of therapy. Some complication like peripheral circulatory failure, Bronchitis, neuropsycheatric manifestation[10], glomerulonephritis and D.I.C. were seen more frequently than traditionally observed. Some rare and unknown complications were also noticed which included hepatic encephalopathy, polyarthritis, pericarditis, spontaneous spleenic rupture[113] and cerebellitis.

Drug resistance

During early period of our observation of multidrug resistants enteric fever, the choice of effective quinolones was limited to Norfloxacin and disc sensitivity reports favouring only quinolones, gentamicin, and cephalosprins, the therapy was difficult especially in view of the severity of disease. Even then successful treatment was achieved by Norfloxacin (400mg BID)[12] and also by a combination of Gentamicin (60mg 8 hourly) and cephalosporin (cephaloxain 2gm/day).

For the convenience of therapy and also being effective Norfloxacin therapy overtook the other group and achieved populavity for multidrug resistant typhoid fever. In some patients cefotaxime (6-8gm/day) was given alone or in combination with gentamicin with fair success.

Subsequent availability of other quinolones lead to use of ciprofloxacin and pefloxacin for effective treatment of drug resistant enteric fever, and more frequently in critically ill patients in view of availability of parental formulations, of such drugs. In certain circumstances (During pregnancy and younger patient) cefotaxime, cefuroxin and ceftrioxone were used without failure.

Out of all the drugs tried quinolones showed a better acceptability though different side effects have been observed with high morbidity[13].

Mortality

A more serious profile of typhoid is currently emerging[11,12,14]. During the last decade there has been conflicting reports about the mortality of these patients and there is an upward trend in case fatality rate (12-18%) in documented, hospitalized adult patients of typhoid[2]. One of the important factors is the emergence of drug resistance strains of salmonella, and also delay in correct diagnosis. The spectrum of typhoid is definitely widening and more serious as compared to that in the past. The picture is dependent on number of factors like socio-economic status, in-adequate treatment, changing clinical presentation, occurrence of complications and emergency of resistance of salmonella thphi. Quinlones are effective drugs for the treatment of thphoid, but the development of resistance to these drugs as lately seen by us puts a question mark to there efficacy and the treatment of enteric fever in future is a matter of great concern.

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"Environmental,Tobacco Smoking & Cancer"

Sheikh Aejaz Aziz, M.D;D.M.(Oncology)

Environmental Tobacco Smoking(ETS) is the combination of the fraction of the exhaled smoke after it has been drawn through the tobacco into the smoker,s mouth (mainstream smoke) and the smoke emitted from a burning tobacco-product between the puffs (sidestream smoke).

ETS is injurious because:

1. Carcinogens present in the mainstream cigarette smoke are present in ETS.

2. ETS is present at levels at which risk would be expected.

3. ETS is absorbed by non-smokers in amounts at which a risk would be expected., and

4. There is increased risk of Lung cancer among the non-smokers with more intensive exposure to ETS (I).

Approximately I/3rd of the Lung cancer among the non-smokers with smokers and about 25% of the Lung cancer in the non-smokers in general population comes from passive exposure to the cigarette smoke (2).

In our valley, especially in the present adverse prevailing conditions and winter, majority of people stay indoors and travel in close, leading to a tremendous exposure to ETS, besides air pollution.

Regular smoking begins during adolescence generally after the age of 12, ETS in contrast, begins at birth (or possibly in-utero). The risk associated is related to the intensity of exposure (number of cigarettes smoked per day) and its duration, but the latter has more powerful effect. Children exposed to parentral cigarette smoke have levels of risk as do the spouses of cigarette smokers. The distribution of the intensity of exposure, however may be more biphasic in children that in adults, with some children receiving heavy exposure and many receiving none. Children who have non-smoking parents (who tend to have non-smoking friends) and siblings who smoke may have few sources of exposure to ETS. Adults, in contrast, have wide variety of exposure sources including work and recreational activities. Exposure of children to ETS is doubly distressing. Children experience the respiratory effects of smoke directly and they begin to accumulate exposure during one of the few periods in life when a smoke free environment might be readily achieved. Children whose parents and siblings smoke may have exposures comparable to those spouses of smokers and will have begun those exposures 20 or more years sooner (3). Discussion of the risks associated with the various levels of exposure often ignore the probability that the exposure of the non-smokers to ETS, while much in the lower intensity that exposure of the non-smokers to mainstream smoke will be 12 or more years longer. The longer exposure may partially offset the lower intensity of the exposure, resulting in the higher risk of the Lung cancer than predicted by single dose-response extrapolation from the doses received by active and passive smoking. ETS increase risk for Lung cancer for females who never smoked and the risk of cancer doubled for female, who reported 40 or more smoke-years of house-hold exposure adulthood. The risk being highest for Non-adeno carcinoma (although there in an increase in Adenocarcinoma group as well (4). Cigarette smoking is a major cause for cancer Lung, Larynx, Oral-cavity, and oesophagus and a contributory factor for Urinary bladder, Pancreas and Kidney. A relation has also been noticed between cancer of Uterine cervix and hematological malignancies. In US, smoking is identified as chief avoidable cause responsible for one million years of potential life lost for US population (5).

Thus the knowledge of the risks stemming from the exposure to ETS makes justification for allowing continued exposure of adults unatenable and the continued exposure of children unthinkable. It would be a public health tragedy, if twenty years in the future, we are obliged to total the costs in human lives and suffering of our failure to act on scientific certainty.

To fortify the spirit of anti-ETS movement, I advocate following measures, which would prove vital to promote the health of the community:

i) Promulgation of an effective anti-ETS legislation, especially at public places including Hospitals and means of travel.
ii) Re-inforcement of 'No-smoking' message at all important places.
iii) Targeting the high risk population for behavioural changes (Females, blue-collar workers, especially children and adolescents).
iv) Health education to evolve a public movement against ETS.
v) Priority package for non-smokers in various government & semi-government institutions.
vi) Abandoning the current trends in Tobacco advertising and marketing.
vii) Motivation and mobilisation of mass awareness against health hazards of smoking in general and ETS in particular.
viii) Active involvement of Physicians and other health-care personnel as effect agent/s of change in the life style and behaviour of the community.

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Editor's Note (News):-

Health ministers recommend ban on tobacco advertisements at PARIS, on Oct.13,(1994). Health ministers from several European and Aisna countries have prescribed a ban on tobacco advertisements at the ninth world conference on tobacco and health as the best remedy for rising death tolls caused by smoking.

Anti-smoking straegies banning tobacco ads, raising cigarette prices, regulating smoking in public places and educating, health ministers said.

"Current trends in treatment of Benign Prostate Hyperplasia (BPH)"

Dr. Ghulam Jeelani, MS, ICS, Dip. Urol (England UK)
Institute of Urology, University of London UK.

Introduction:

Benign porstatic hypertrophy (BHP) is the major urological problem affecting men above age of 50, producing voiding dys function and symptoms of prostatism. Until recently transuretheral resection of obstructing prostate (TURP) was the "Gold standard" of definitive treatment. Indeed all newer modalities of treatment will have to be compared to this as far as the results are concerned. Last few years have witnessed newer innovations in the treatment of BPH due to better understanding of patho-physiology of this disease and due to technico-logical advancements.

A. ***Pharmaco Therapy***:
Medical treatment offers sympatomatic relief from distressing symptoms of prostatism and is best suited for patients either unfit for surgery or awaiting definitive treatment.

- i) ***a-blockers***:- Presence of a and ß adrenergic receptors in lower urinary tract and their role in voiding have led to development of a-adrenergic blockers like phenoxybenzyum (Dibenzylin), prazosine and terazosine. They have been shown to decrease the outflow resistance in the prostatic urethera.
- ii) Finstride (Proscar): 5- a -reductase inhibitor works by actually reducing the size of prostatic gland, by blocking the conversion of testestrone to dihydrotestestrone (DHT), the latter is necessary for the grown of prostatic gland. The enzyme 5-a-reductase being necessary for this conversion.

B) ***Intraprostatic Stents***:
Biocompatible, intra prostatic stents (urolome, memokath) mode of non-irritant alloy of Nickel and Titanium with a shape memory is inserted into the prostatic urethra endoscopically by transuretheral route. Over a period of time actually urothelium grows over the stent and that holds apart the prostatic urethera thus relieving obstruction. Stent can be removed endoscopically if necessary. Again this modality of treatment is best suited for patients unfit for surgery.

C) ***Thermotherapy***:
Transuretheral microwave thermotherapy (TUMT) using hyperthermia to about 45°c for an hour or so the heat delivered via a catheter generated from a microwave generating machine. The procedure is simple, done under local anaesthesia as a day case with no hospital stay and minimal morbidity. The heat delivered results in prostate gland ablation with the resultant improvement in outflow obstruction.

D) ***Endoscopic Laser Ablation (ELAP):***
Laser energy is being used to ablate the prostate gland. The energy is delivered by transuretheral route using Neodium Yag lassers utilizing either non contact side firing delivery system (prolase) or by interstitial contact coagulation (ICC), ultasounds are also used to guide the actual site of energy delivery or else the same can be achieved endoscopically. This treatment modality again has the advantage of minimal morbidity done as a day care under local anaesthesia.

E) ***Balloon Dilatation of prostate (BDP):]***
Inflatable balloon mounted on a catheter mechanism are introduced into prostatic urethera and dilatation done upto a certain atmospheric pressure. The resultant dilatation of the obstructed prostatic urethera secondary to BHP gives rise to symptomatic relief and improvement in voiding function. The procedure is repeatable and minimally invasive. Its main dis-advantage is that the effect of dilation is temporary.

F) ***Bladder Neck Incision***:
Transuretheral incision of prostate (TUIP) as against TURP by incising the bladder neck at 5 and 7 o-clock position across the obstructing prostatic lobes results in relief of outflow obstructing symptoms comparable to results in relief of outflow obstructing symptoms comparable to results of TURP. The procedure is minimally invasive with minimal morbidity and mortality.

The above mentioned minimally invasive procedures are extensively tried in developed countries and long term follow up is being assessed. Since the natural history of prostatic enlargement is unpredictable, it is difficult to say at this time the long term efficacy of these procedures. These options are attractive because of being minimally invasive with low morbidity, negligible mortality and cost effective. Ultimately the results of these procedures will have to be compared to the "Gold stand" therapy of prostate enlargement i.e. TURP.

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Incidence & Pattern of Infections in Diabetes Mellitus

A Retrospective Study

*Dr.Abdul Hamid Zargar[*], Dr.Shariq, Rashid Masoodi[**] , Dr.Bashir Ahmed Laway[***],
Dr.Mohd. Amin Akhter[****], Dr.Fayaz Ahmed Bhat[*]*

Summary

The case record of 792 patients with diabetes mellitus admitted in last 5 years were reviewed for incidence and pattern of infections. All patients had been treated al Institute of Medical Sciences, Srinagar-Kashmir. Analysis of data revealed presence of infection in 154 (19.4%) diabetics. There was no effect of age, sex or type of diabetes on presence on severity of infection. Five most common infections encountered were urinary tract infection (28.6%), tuberculosis (20.1%), skin and soft tissue infections (14.3%), bacterial plneumonia (10.4%), and foot infections (10.4%). Among the major chronic diabetic complications peripheral neuropathy was seen in 78 (50.6%), retinopathy in 18(11.7%), and nephropathy in 22(14.3%) patients with infections. We conclude that infections continue to be an important cause of concern in diabetics in developing countries, and measures should be taken for prompt recognition and treatment of infections in diabetics to reduce the associated mortality and morbidity.

Introduction

Infections are believed to occur with increased frequency in patients with diabetes mellitus, whether or not this is true, remains controversial[1] Nolar et al in 1978 reported that granulocytes from ambulatory hospitalized diabeties with fasting blood glucose values of>200mg per deciliter had an impaired ability to engulf and kill opsonized staphylococci. The granulocyte function improved after more intensive management of diabetes mellitus[2]. Infection is associated with impaired carbohydrate tolerance[3-5] and contributes to approximately 25 percent of the deaths associated with diabetic ketoacidosis[6-8].

Prior to the advent of modern anti-microbial therapy, infections accounted for much of mortality in diabetes. With the advent of anti-microbial agents fewer deaths occur due to infections in developed countries. However, in developing countries infections in diabetics continue to be a major problem. In this retrospective study we attempted to find out the incidence and pattern of infections in patients with diabetes mellitus who were admitted in the department of Endocrinology, Institute of Medical Sciences, (IMS) Srinagar, Kashmir from January,1987 to March, 1992. This hospital is a 500 bed referral centre, with patients coming from all over the Valley of Kashmir.

Material and Methods

Hospital records of all patients of diabetes mellitus who were admitted in the Department of Endocrinology, IMS, Srinagar, Kashmir from January, 1987 to March, 1992 were reviewed. All available data for each patient was studied in detail to determine the incidence and clinical profile of infections in these patients. These patients were mainly studied for age and sex distribution, type and duration of diabetes, presence of complications; and the presence and nature of infections.

Results

We could retrieve the records of 729 patients with diabetes mellitus who were admitted in the Department of Endocrinology, IMS, Srinagar from January, 1987 to March, 1992. Out of these 792 patients 154 (19.4 percent) had evidence of concomitant infections constituting about one fifth of all diabetic population (Table-1).

There was no significant difference in the age and sex distribution of total diabetics and diabetics with infection. Out of 792 patients 122 had IDDM, 642 had NIDDM, 8 had fibrocalculous pancreatic diabetes and in 20 patients records were not clear about the type of diabetes. There was no significant difference in the frequency of infection in patients with IDDM or NIDDM (Table-II). The median duration of diabetes in patients with infections was 2 years in type-I diabetes mellitus and 5 years in type-II diabetes mellitus. Five most common infections encountered in patients with diabetes mellitus were urinary tract infection (28.6%), tuberculosis (20.1%) skin and soft tissue infections (14.3%) bacterial pneumonias (10.49%) and foot infections (10.4%); all these constituting about eighty percent of all infections (Table-III). Among the major chronic diabetic complications in patients with infection peripheral neuropathy was seen in 78 (50.6%), retinopathy in 18 (11.7%) and nephropathy in 22 (14.3%) patients, Among the miscellaneous infections recorded were periodontitis, vulvulitis, chronic suppurative otitis media, mucormycosis, balanitis, amoebic hepatitis, septic arthritis and osteomyelitis.

Discussion

Infections in diabetic patients continue to be a major cause of morbidity ad mortality. Even though the incidence of infections in diabetics in the developed countries has steeply gone down[9], the same does not seem to be true for developing countries like ours as this study shows.

In our study out of 154 diabetics with infection 44 (28.6%) had urinary tract infection, majority (68.2%) of whom were asymptomatic. E-Coli was the commonest causative organism. Urinary tract is probably the most common site of infection in diabetics. Diabetics have been found at autopsy to have five-fold greater frequency of acute pyelonephritis than non-diabetics[10]. Although most urinary tract infections in diabetics are asymptomatic[11-13], the presence of diabetes predisposes to more severe infection[14]. In one study E-Coli was the commonest cause of emphysematous pselonephritis (50%) while the remainder being caused by other gram negative bacilli[15]. None of our patients with UTI had evidence of emphysematous pyelonephritis.

Tuberculosis was once a common and serious problem in diabetic patients. In 1930's tuberculosis was 3-16 times more frequent in diabetics than non diabetics[16-18]. In our study 31 out of 154 (20.1%) patients had tuberculosis. In these patients diagnosis of tuberculosis was done on radiological grounds, acid fast bacillus (AFB) positivity in sputum and on culture studies. Currently tuberculosis is less of a problem in diabetic patients, however diabetes may still predispose to reactivation of tuberculosis[19]. All these patients of tuberculosis were treated with conventional antitubercular regiments as tuberculous chemotherapy is reported to be equally effective in diabetic and non diabetic population[20].

Skin and soft tissue infections were found in 22 out of 154 patients of diabetes mellitus with infections, and in half of these cases culture had grown staphylococcus. There is a general belief that diabetic patients are predisposed to staphylococcal skin infection and the results of a study in recent years indicated that staphylococcal colonization correlated with poor glycemic control[21] however diabetes predisposes to staphylococcal skin infection has not been proved. Gram negative wound infections are said to be three times more frequent in diabetics than in non diabetic individuals[22].

Bacterial pneumonia remains important infection in diabetic patients and is frequently caused by Gram-negative organisms or staphylococci[23]. However, diabetes does not appear to predispose to pneumococcal pneumonia[24]. In our study 10.4 percent of diabetic patients with infection were diagnosed to have bacterial pneumonia. Most of the times diagnosis was made on clinical and/or radiological grounds. Why diabetic patients should be predisposed to Gram-negative or staphylococcal pneumonia is not clear.

Foot infections are common and serious problems in diabetic patients. Infection may develop in the toe nail bed, beneath planter calluses or in ischemic or neuropathic ulcers. Once established these infections may extend to involve deeper soft tissues, joints or bones[25]. In this study sixteen out of 154 diabetics with infections had primarily to be admitted for foot infection, half of which needed amputation. Most of these patients had a prolonged hospital stay. Other infections recorded were upper respiratory infections, pyrexia of unknown origin periodontitis, vulvulitis, chronic suppurative otitis media, mucormycosis, fungal infection, balanitis, amoebic hepatitis septic arthritis and osteomycletis.

Although morbidity and mortality in diabetics due to infection is becoming less important in developed countries, in developing countries these still constitute an important cause of concern. Measures should be taken in diabetics including care of foot, prompt recognition and treatment of an early infection giving specific antimicrobial agent for a sufficient time and maintenance of an euglycemic state, usually with insulin therapy till the infection is eradicated.

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Table-I:

Age and sex distribution of diabetics with infections

with diabetes	No. of patients with infections	No. of diabetics
Total No.of patients	792	154(19.4%)
Males	410	76
Females	382	78
Mean age (Years)	46.4	47.2
±SD	±14.2	±12.1

Table-II
Incidence of infections in patients with different types of diabetes

	No.of patients with IDDM	No.of patients with NIDDM	Miscellaneous
Patients with infections (n = 154)	20	134	0
Patients without infections (n = 638)	102	518	28

p>25 (Not significant)

* Miscelllaneous = Patients with pancreatic diabetes and other where type of diabetes was not clear.

Table-III
Incidence of infections

S. No.	Type of infection	No.of patients	(%age) (n = 154)
01.	Urinary tract infection	44	(28.6)
02.	Tuberculosis	31	(20.1)
03.	Skin and soft tissue infections	22	(14.3)
04.	Bacterial pneumonia	16	(10.4)
05.	Foot infections	16	(10.4)
06.	Upper respiratory tract infections	13	(8.4)
07.	Pyrexia unknown origin	09	(5.8)
08.	Choleystitis	05	(3.2)
09.	Chronic suppurative otitis media	02	(1.3)
10.	Others	14	(9.1)

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** Out of 154 diabetics with infection, some had more than one infection present concomitantly making total number of infections 171.*

Myelodysplsia and Preleukemic Syndromes: (MDS/PLS)

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Myelodysplasia are a group of clonal disorders of pleuri-potential cells characterised by dysmyelopoiesis and peripheral blood cytopenias.

Myelodysplasia may be primary or secondary. The latter is seen secondary to chemotherapy and/or Radiotherapy and exposure to toxins like benzones.

a) Primary form: is classified by French American British (FAB) collaborators into five subtypes. Modified FAB classification has added a few more subjects.

Sub-types include:-

1. Refractory anaemia (RA)
2. Refractory anaemia with ring sideroblasts (RARS)
3. Refractory anaemia with excess blasts (RAEB)
4. Refractory anaemia with excess blasts in transformation (RAEBIT)
5. Chronic myelo-monocytic leukemia (CMML)
6. Chronic myelo-monocytic leukemia in transformation (CMMLIT)
7. 5q-Syndrome

Preleukemic snydromes (PLS) has also been defined as clonal proliferation of pleuripotential stem cells, but with no leukemic blast cells in peripheral blood or bone marrow in beginning. Several conditions have been included in PLS syndrome namely.

1. Haemopoietic dysplasia
2. A plastic anaemia
3. P.N.H.
4. Red cells aplasia
5. Conenital amega-karyocytic thrombocytopenia
6. Chronic idiopathic neutropenia.

Haemoporetic dysplasia: is characterised by cytopenias, hypercellular marrow, dyshaemopoiesia and high potential for acute leukemic transformation. Therefore it merges with MDS/PLS syndrome since both share dyshaemoporesia in addition to variable potential for ANLL.

It is well accepted that approximately one-third of ANLL in adults and one-tenth of ANLL an preceded by a smoldering course of MDS/PLS.

Etiology of primary dysplasis/PLS

It is not known, two factors seen to play a role:

1. ***Age:*** Nearly three-fourth of cases are seen in the age fifty and over.
2. Cellular oncogene dysfunction is suspected. Point mutation in codons 12,13,59, or 61 in the N-ras, K-ras or H-ras have been described in a minority of cases.

Pathogenesis: MDS/PLS, as mentioned above are clonal proliferation haemopoietic stem cells, which gradually replace the normal haemopoietic cells. Clone as it expands, loses its capability to differen-tiate, with maturation arrest, dyshaemopoiesis with ineffective haempoiesis and qualitative abnormalities. When immature cells assume a mass of $1x10^{12}$ cells, it is identified as acute leukemia.

Case study: A brief data of 8 cases of MDS/PLS is presented here.

Case NO.1: *62M Peripheral Blood. Refractory macrocytic anaemic, luecopenia, with poor lobulation, and cytoplasmic granularity of neutrophils. Thrombocytopenia, with giant platelets and absence of platelet aggregate in peripheral blood.*

Bone Marrow: Hypercellular with erythroid hyperplasia. Megaloblastoid erythropoisis, maturation arrest and nuclear fragmentation Iron stores (Grade III) Pathological sideroblasts presents, with a few ring sideroblasts. Granlocytic hypoplastic with abnormal granulation (few and abnormal shape) Blast cells-3%.

Megakaryocytic: Reduced in number with poor nuclear lobulation, cytoplasive granularity and platelet production.

Diagnosis: Myelocysplasia (R.A)

Clinic course: Progressive pancytopenic and death related to severe a pancytopenias within 1½ years.

Case No.2: *Diagnosis: Myeloidyspasic (Refractory anaemia virtually) identical to case 1, lost to follow up.*

Case No. 3: ***70M :*** *Severe pancytopenia, with Hypercellular marrow, maturation arrest and dyshaemopoiesis.*

Diagnosis: Haematopoietic dysplasic (Preleukemia syndrome)

Cause of death: Severe progressive cytopenia.

A group of refractory anaemia

Case No.4, 68M Pancytopenia with trilineage dyshaemopoiesis
Case No.5, 60M (Case No.4, diagnosed initially in Saudi Arabia).
Case No.6, 52M

Managed symptomatically

Under follow-up, 3 months, Case No.4 has a total follow up of over 2 years.

Case No.7, 37M Pancytopenia with trillineage dyshaempoiesis.

Blasts (Type, I, and Type II) 22% in B.M. aspiration smears.

Cluster of blast cells in trephine biopsy: Iron stores

Diagnosis : Myelodyeplasia (RAEBIT)
Management: Symptomatic : Under observation 4 months.

Case No.8, 33M: *Cytopenia with trilineage dyshaempoiesis and progressive over a period of 18 months with transformation to AML.*

Discussion : A study of eight cases of MDS/PLS presented here comprise of 5 cases of refractory anaemia, one case of haemopoietic dysplasia and one case of refractory anaemia with excess of blast in transformation and one cases of acute leukemic transformation. All the cases were males and all but two were aged over fifty. Literature reports only slight male predominance. There was no history of obvious exposure to chemotherapy, radiation or toxic agents. By profession two are retired officers, two business, two farmers and one employed in Public Health Engineering.

Clinical data were conspicious by their absence except those related to anaemic, leuopemia or thrombocytopenia.

The term myelodysplasic and pre-leukemie embrasses various sub-types of myelodysplasic and haemopoistic dysplasia referred to earlier. Dyshaemopoiesia is an essential feature, but acute leukemic transformation is variable with each sub-type. For example it has been reported in 5-10% cases of RA RARS and 5q syndrome and 20-60% in RAEB, RAEBIT and haemopoietic dysplasia. Other major killer are cytopenias. Two of our cases died from severe cytopenias and one from AML.

Differential diagnosis: includes megaloblastic anaemia, aplastic anaemia and pancytopenia with hypercellular marrow associated with nonhaemopoietic cancers (Para-neoplstic syndrome).

In Megalobastic: anaemic, mild dyserythropoiesis and response to Folate/B12 settles the issue. Aplastic anaemia occasionally may show hypercellular marrow with pancytopenia. Hypercellularity is patchy and dysmyelopoiesis is not a feature.

Paraneoplastic syndrome: with hypercellular marrow is not associated with dysmyelopoiesis. Further, clinical data, helps to settle the tissue.

Diagnosis: Diagnosis is made on the basis of clinical data haematologic findings and cytogenetic studies.

Clinical data: In addition to anaemia, infection and thrombocytopenis, occasionally patients are known to present in addition with arthritis and rarely with diabetes insipidous and neutrophilic dermatoses. Mild hepatosplenomagely is seen in 5-10% or upto 40% in CMML.

Haematologic data : include peripheral blood and bone marrow smears/trephine biopsies.

In summary findings are those of dysmyelopoiesis

(For details-seen any T.B. of haematology).

The most common and essential to diagnosis of MDS/PLS is dyserythropoiesis. Dysgranulomonocytopoiesis is an important feature. Dysmegakaryo poiesis is present in many form.

Cytogenetic studies: are not essential for diagnosis but are of prognostic value, since those cases of MDS/PLS associated with chromosonal anomalis are associated with high risk of leukemic transformation.

Chromosonal abnormalities are in general like those of ANLL with some exceptions like t (8:21) of M2 and t(15:17) of M3, but are seen in lower frequency than ANLL.

Treatment:

1. In general is symptomatic and conservation and includes vit.B_{12}, Folate, suitable antibiotics, and steroids.
2. Other agents that have been tried include erythropoietin colony stimulating factors, Cis-retinoisis etc.
3. Patients aged 70 and over are given low doses of cytosine arabinoside.
4. Patients 40 and under especially those with excess blast cells (consider by some as AML-M2) are considered for a bone marrow transplantation and 2) given low doses of chemotherapy including daunomycin, cytosine arablinoside and 6-thio-guanetic.

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"Aetiological study of vertigo in Kashmir”

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Vertigo is a sensation of rotation, either of the individual or of his environment, resulting from stimulation of the labyrinth or its central connections.

Diagnosis as to the etiology of vertigo remains a problem to the Otolaryngologist as a wide variety of disorders can present with vertigo.

The causes can be divided into two main groups:

1. Central
2. Peripheral

Central Causes: are those causes where central connections of vestibular nerve are involved.

1. Epilepsy
2. Disseminated sclerosis
3. Posterior fossa tumour
4. Alcohol intoxication
5. Cerebrovascular diseases: This includes;
    i. Thrombosis of posterior inferior cereblelar artery and internal auditory artery.
    ii. Compression of vertebral artery due to neck rotation and bony spur.
    iii. Basilar insufficiency
    iv. Cerebellar haemorrhage

Peripheral causes include the following:

1. Menier's disease
2. Vestibular neuronitis
3. Infective labyrinthitis
4. Toxic labyrinthitis
5. Positional vertigo
6. Metabolic disease ....diabetes
   hypothyroidism
7. Miscellaneous:
   Motion sickness
   -Eustachian tube dysfunction
   -Sinus infection
   -Dental infection

Material and Methods

We have tried to find out the various causes of vertigo in our set up amongst patients presenting at S.M.H.S. Hospital, Srinagar, Kashmir. 200 cases presenting with the symptom of vertigo were seen during a two year period from 1985 to 1987. A detailed history was taken and E.N.T. check up was done. The cardiovascular and neurological check up was also done wherever indicated. Audiological investigations included audiometry, caloric test and positional test. Radiological examination of the skull, paranasal sinusis and cervical spine were done. Blood sugar and serum cholesterol estimation and VDRL was done wherever indicated. It was attempted to arrive at an etioilogical diagnosis of these causes.

Results

Two hundred patients presenting with the chief complaint of vertigo were taken up for this study. 75% of the patients were in the age group of 20 to 50 years. Sexwise there was male preqonderance, ratio being M:F. 3:2.

Table-I

Age and sex distribution

Age	Male	Female	Total
0-10	-	-	-
11-20	7	5	12
21-20	25	18	43
31-40	28	22	50
41-50	32	24	56
51-60	20	11	31
above 60	8	-	8
	120	80	200

Causes of vertigo

Diagnosis as to the etiology of vertigo could be arrived at in 138 cases out of the total of 200

(69%). Table No.1 shows the causes of vertigo seen in our set up. Definite diagnosis as to the cause of vertigo could not be arrived at in 62 cases (31%).

Table No.2
Causes of Vertigo

Disease	No.of Cases	%tage
-Vertibro basilar insufficiency	47	23.5
-Benign positional vertigo	24	12
-Menier's	18	9
-Vestibular neuronitis	13	6.5
-Infective labyrinthitis	12	6
-Anaemia	7	3.5
-Otosclerosis	4	2
-Acoustic Deuroma	4	2
-Streptomycin ototoxicity	4	2
-Floating foot plato	2	1
-Perilymph fistula	2	1
-Heart block	1	0.5
-Unclassified	62	31
Total	**200**	

Discussion

- Vertebro-basilar insufficiency secondary to Cervical spondylosis, atherosclerosis and hypertension was the commonest cause of vertigo (23.5%) in our population as both these disease survival spondylosis and hypertension are quite common here. Similarly Bhatia and Deka (1985) found this etiology in 18.6% of their cases of vertigo.

Benign paroxysmal positional vertigo was the second most common cause (12%). 75% of these had either history of head injury of evidence of a past otitis media.

Vertibro-basilar insufficiency and benign paroxysmal positional vertigo accounted for about one-third of the cases; while in one-third no diagnosis as to the etiology could be arrived at. All the other causes accounted for one third of the cases.

In our population Menier's disease accounted for only 9% of the cases while in Western literature 60-70% of cases of vertigo have been found to be due to Menier's disease (Cawthorne T, Griffith A 1961, Mawson S.R. & Ludman H. 1979).

Amongst the streptomycin group, one patient got severe vertigo with vomiting just after one injection of streptomycin. His renal functions were normal.

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Fertility pattern in the field practice area of

Social and Preventive Medicine Department

Government Medical College Srinagar.

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Abstract:

This community based study had been carried out in the field-practice area of Government Medical College, Srinagar and revealed CBR = 31.58 and CDR = 9.54/1000 population. The mean age at marriage, mean age at first delivery, GFR, TFR, GRR, rate of natural increase and age-sex adjusted birth rates /1000 population were observed to be 18 years, 19.41 years, 134.46, 3.52, 1.71, 2.2% and 30.20 respectively.

Keywords: Fertility, ASFR'S, GFR, TFR & GRR.

Introduction

As early as 1980's the Government of India stressed the imperative need to stabilise country's population. However, no concrete efforts were mode to realise the Goal. In fact, we are still for from achieving the Goal of a crude birth rate (CBR of 25/1000 population which was supposed to be reached by 1973, Ist alone the Goal of stablising the population size. The working group on population constituted by the planning commission in 1978, recommended a long term demographic goal of attaining NRR = 1 by 1996, now to be achieved by 2006-11 as per the seventh five year plan. In view of the urgency of achieving NRR = 1 in India an attempt has been made in this paper to study various fertility indicators and factors in this part of country to give a base-line information for the future planning and evaluation of different programmes in this regard.

Material and Method

The present study had been conducted for a period of one year i.e w.e.f. 1.4.88 to 31.3.89 in Hazratbal, Ganderbal and Lal blocks which form the field practice area of Govt. Medical College, Srinagar. Hazratbal, Ganderbal and Lar are Urban, Semi-urban and rural blocks with a population of 92, 067, 49,157 and 39,277 respectively. On the basis of simple random sampling procedure 30 villages, 10 villages from each block were selected for the study centering 30, 809 (17.07%) of the total population -12,403 (13.48%), 8845 (17.99%) and 9561(24.34%) from Hazratbal, Ganderbal and Lar respectively. The information was collected on the predesigned and protested schedule covering socio-demographic characteristics births and deaths during the study period in the sampled villages by bi-weekly home visits.

Results

The crude birth rate in the study area was 31.58/1000 population Table-I. The crude death rate and the rate of natural increase were 9.54/1000 population and 2.20% respectively of the total births occuring in the three blocks during the study period 27.83% were of the Ist birth order Table-II. The table depicts a dedlining tendency till the 4th birth order after which an increase (14.47%) is seen in the plus 5th birth order. There were 5036 children in the age group of 0-6 years thus giving a child woman ratio of 699.3/1000 woman in the reproductive age group. Analysis of age specific fertility rates (ASFRS) in different age groups show the rate to be 131.29 in the 15.19 years age group increasing to 280-84 in 20-24 years age group. Thereafter it shows a decline being 3.60 in 40-44 years age group. The sex-age adjusted birth rate (SAABR) of the population had been founded to be 30.20 per thousand women in the various five year age group from 15-44 years. Table-IV depicts general fertility rate (GRR) as 134.46, 3.52 and 1.71 respectively during the study period. The mean age at marriage and the mean age at first delivery were 18 and 19.41 years respectively for those women who delivered during the study period in the sampled villages.

Discussion

The most frequently quoted fertility rate is CBR which measure the overall effect of fertility on the current growth rate of the population. The CBR of 31.58/1000 population was lower than the national figure of 52.5/1000 for 1986 (Health information India, 1988) (Table-I). The natural rate of increase of 2.20% observed during the study is slightly higher than 2.13% reported for India for 1984, keeping in view the goal of growth rate of 1.66 by 1990, efforts to promote family planning methods need to be sustained. 53.33% of the births in the present study were of Ist and 2nd birth orders and 47.67% of the 3rd and above birth orders. This is due to the effect of reduced infant and early childhood mortality and changing pattern of the family size desired by the parents thus leading to low percentage of births in 3rd and above birth orders. Mukerji, 1987. (3) had reported 63% of births in 3rd and above birth orders in India (Table III). The child woman ration of 699.3/1000 woman in our study was less than 830.6/1000 woman as reported by Bhatnagar et al (4).

By providing the clear picture of risk of child bearing in a population A SF Rs allow to study changes in fertility and fertility differences between population. the maximum ASFR, 280.84 had been observed in the age group of 20-24 years, whereas Ramachandran, V.1987(5) reported highest ASFR,302.1, in 15-19 years age group followed by 263.1 in 20-24 years age group. This indicates a favourable trend in our three blocks. To reduce the effect of age-sex composition of the population on crude birth rate, the sex age adjusted birth rate 30.20 was calculated and was found to be higher (6) than reported by Ramachanderan (5).

The growth of human population depends upon fertility, the observed TFR (3.52) GRR (1.71) and GFR (134.46) in the present study are lower than national figures of 4.51, 2.20 and 136.0 respectively for 1980-85. The lower figures of fertility indicates in the current study reflect a favourable thrust in our blocks in the context of achieving the targets of family welfare programme by 2000 AD.

The age at marriage and first delivery has a bearing on fertility pattern. Many attempts have been made during the past 50 years through social legislation to influence the teen-age marriage pattern in India. The 1978 Act revised the minimum age at marriage for females to 18 years. The mean age of marriage in the present study had been observed to be 18 years in the three blocks. This is higher than the national figure of 16.7 years Registrar General, 1989 (6). The mean age at first delivery had been found to be 19.41 years which is higher than the study conducted by Prakasm (1980) in Bombay (7) Table IV.

Table-I

Distribution of live Births in3 Blocks Burning Study Periods

Block	Live Births M	F	T	Crude Birth Rate(CBR) Per 1000 Population
Hazratbal	174	161	335	27.01
Ganderbal	147	149	296	23.45
Lar	164	178	342	35.77
Total	485	488	973	31.58

Table-II

Distribution of Births in three Blocks Birth Order during Study Period

Birth order	Births Hazratbal	Ganderbal	Lar	Total
Ist	100 (29.15)	82 (27.52)	93 (26.80)	275 (27.83)
2nd	92 (26.82)	70 (23.49)	80 (23.06)	242 (24.50)
3rd	78 (22.74)	57 (19.13)	80 (23.06)	215 (21.77)
4th	33 (9.62)	34 (11.40)	46 (13.25)	113 (11.43)
5th	40 (11.67)	55 (18.46)	48 (13.83)	143 (14.47)
Total	343 (100.0)	298 (100.0)	347 (100.0)	988 (100.0)

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Syncope

Dr.Mir Mohammed Ashraf, MRCP FRCCP.

I though to start with the Syncope which is very common in Kashmir. Syncope or fainting attack is a transient loss or consciousness and it is mostly due to cerebral hypoxia secondary to inadequate cerebral blood flow. It must be distinguished both from conditions in which there may be weakness, faintness or dizziness, but no loss of consciousness and from other conditions such as epilepsy, in which patient actually loses consciousness. The following conditions may be confused with the 'Syncope':

Vertigo

Causes the patient to experience sense of movement. The patient feels that he is moving or the objects are moving around him. The patient will fall on the ground, but he does not lose his consciousness and his pulse and blood pressure remains normal.

Sleep

An elderly patient may dose briefly in a chair, simulating Syncope, but the pallor and pulse changes of Syncope are absent. Sudden appearance of such dozing off episodes may be a sign of Syncope.

Narcolepsy

Refers to parosysmal attack of sleep without loss of consciousness. Many patients with narcolepsy also have cataplexy, sudden attack during which muscle tone is lost and patient may fall to the ground.

Conversion Hysteria

The patient usually a female, may have an attack of hysterical fainting. However, there is no pallor, and there are changes in pulse or blood pressure. The fall to the ground is characteristically graceful causing no injury.

Hyperventilating Syndrome

Hyperventilation will lead to actual Syncope. The patient compalins of dizziness and Tight-headedness. He may note circumoral paresthenia, numbness and tingling with fingers and toes, sweating, palpitation, tiwits and tremulousness, dyspnae, feeling of fear and uneasily tighthers around the chest, nauxia and vomiting, diarrhoea, blurred vision and even loss of voice may occur.

Hypoglycemia

Occurring spontaneously or after an insulin overdose may produce Syncope or even coma. It may cause weakness, trembling, diaphoresis and other symptoms palpitation but the blood pressure is normal.

Convulsive disorders

May stimulate syncope because convulsion may occur during Syncope if unconsciousness lasts approximately ten second or longer. In epiteptiform attack, however, the convulsions produce the loss of consciousness. Proceeds the convulsions-tonic convulsine movements with the patient's eyes upwards are coming in epileptiform attacks but are rare in Syncope. Chromic attacks may occur in born conditions, Tongue felling and urinary incontinuance are also common in epileptic seizures, but are rare in Syncope. Patient in Syncope may feel weak but are mentally alert. In petitmal seizures, there is transient loss of consciousness, but the patient does not fall down. Another due to unconsciousness due to a seizure as the presence of metabolic acidosis with an abnormal large anion gap. Syncope can be classified in principal ways as:-

1. Simple Syncope
2. Syncope of cardiac origin,
3. Syncope due to postural hypotension,

4. Syncope due to excessive vegal reflexes.
5. Syncope due to cerebral factors.

Simple Syncope

Precipitated by (i) stress (ii) initiated when the subject is standing or sitting. When this subject becomes recumbent, sudden pain fright, vernifunction the sight of blood. The patient may faint without having or may experience a brief period of premonitoring symptoms including nausia, pallor abdominal discomfort, weakness, perspiration, unsteadiness and blurred vision-Syncope occurs when the systotic blood pressure is 70mm Hg, Pulse is 80/mt even 40 beats per minute, consciousness, returns when the patient falls to ground. Syncope of cardiac origin. It may occur in any position. It may occur in recumbent position. The most common cause of Syncope is Bradyarrhythmia and Techarrythmias, complete atriovent-ricular block and Adems stokes Syndrome Aortic stenosis Idiopathie hypertrophy sub-aortic stenosis. During this period there is increased venous return to the heart. It may occur in Mitral stenosis and pulmonary stenosis. Acute Myocerdial infraction and acute pulmonary embolisin, Disseeeting aneurysion of Aorta, Primary Pulmonary hypertension tetrology of fellots, pronlonged QT interval and large 'U' Waves, cardiac tremors or bell value thrombosis, Pace maker Syncope: can occur in a patient with a ventricular demand pacemaker when this patient is spontaneous, heart rate suddenly becomes slow and the Pace-maker starts to function at a slow rate. It is not serious. Patient should be informed about it.

Syncope due to postural hypotension. When the normal person stands up from supine position, there is an initial drop in blood pressure due to pooling of blood with legs. This fall in blood pressure stimulates baroreceptors and a sympathetically mediated vasoconstriction and tachycardia occurs. Diastotic blood pressure may become slightly elevated and systolic blood pressure may rise or fall slightly. It can be classified as either functional or organic.

Functional Postural Hypotension

When normal person stands a tachyeardia initial rise in diastolic pressure occurs, however, such facts as excessive cooling during drug effect or inadequate circulatory volume may interfere with the normal homostatic mechanism when patient falls because of inadequate venous return patient will complain of weakness nausia, pallor or standing and Syncope may occur. It may occur in Athletes, pregnant woman in patient during treatment of angina and nitrates, excessive diauritics, vomiting, dirrhoea, Addison's disease, drugs, ganglion blocking agents, Ismalin, Phenoehiazans, quinidine, ECG may reveal sinus tachycardia and T inverted in II, III AVL.

Organic P:ostural Hypotension

It is due to impaired or absent vesomotor reflexes(!) Idopathic (2) Chr. Postural hypotential Tabes diosach's intercranial tension, multiple cerebral infarton, Simond's diseases, Guillem Bar Syndrome, Diabetic, Neuropathic, Porphyria, Addison's disease, Amyloidosis.

Syncope due to exercise.

Cardiac Sinus syndrome. Hyperventilating of carotid sinus is a relating rare cause of syncope Most patients are older men and may occur in turning of the head, a tight collar or shaving the neck. This can be blocked by Atroine.

Carorid Sinus Sincope cerebral have been described in which unconsciousness occurs without the cardiac slowingor a fall in blood pressure.

Cough Syncope

Occurs during proxym of violent coughing or laughter is due to valsalva effect.

Michturating Syncope

Oesophagal diverticular, Oesophagyl spasm and Gall bladder colic. Glassopharyngeal neuralgia Syncope, fainting like voluntary Syncope can be produced if one has the subject hyperventilating for about a minute and then manually compresses his chest. It is due to valsalva effect.

Syncope due to cerebral factors:

Cerebral factor, metabolic Acidosis, Severe diuresis.

Treatment

Regardless of the type of the Syncope, the following emergency treatment should given.

1. Immediately place the patient feet up on a bed or on the floor. A fallen patient should left with the face up.
2. Lift the lower extremities for 15 seconds to increase oardiac blood return.
3. Loosen the patient's cellar or any tight clothing
4. Check the patient's pulse and blood pressure;
5. If consciousness does not return immediately, extend the patient's neck and raise the chin to prevent the tongue from falling backwards and blocking the airway.
6. When patient regains consciousness, slowly raise him to sitting position and then slowly to standing position, if the patient is raised quickly fainting may occur. it may take as long as 30 mts before the patient is able to stand again.

Definitive treatment depends upon the cause of Syncope; for example hypotensive, hypotensive, hyperventilating and hypoglycemia may occur together, with Syncope due to Aortic stenosis, also with maturating Syncope. Tachy-brady arrthmia will require the medicine. Heart block will need pace maker implantation. Epilepsy will also need the specific therapy.

Prophylasis

Most patients with Syncope will benefit from supporting means. These supports should be put on before the patient gets out of bed. Women should be advised not to wear elastic panty girdles. These girdles extend 6 to 10 inches along the thighs and may act like tourniquets preventing venous return from the extremities and also pulse producing oedema. Pt who suffers from Syncope should be advised to take showers and not to take hot bath, when one steps out of the bath tub after a hot bath pooling of blood with lower extremities may occur leading to Syncope.

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"The Causes & Prevention of Coronary Heart Disease"

Dr.G.R.Nahami, Shefield (U.K); M.B.B.S.D.P.P

Coronary heart disease is the most common single cause of premature death in the West, and a major cause of disability and distress. The prevention of coronary disease is a priority in every country. It is widely recognised that there are a number of measures which, if applied, could reduce the burden of this disease on the community. Unfortunately there is a widespread public belief that there are fundamental disagreements amongst the experts about the causes of coronary disease and, in particular, the role of dietary fats and blood cholesterol. There are certainly differences of option, but most of the important facts are agreed, and so are many of the actions that should be taken to reduce the risk of the disease.

The enormous differences in the incidence of coronary disease between countries and between different parts of the same country, and the remarkable changes in coronary death rates in certain countries over a relatively short time period suggest that environmental factors play a crucial role. If these factors could be identified and corrected, there should be a good prospect of appreciably reducing the incidence of the disease, although it is recognised that genetic factors are also important.

Studies of large populations have shown that certain so-called 'risk factors' are associated with the subsequent development of coronary disease. The best known of these are cigarette smoking, raised blood cholestrol levels, and high blood pressure, but some patients with corobary disease do not smoke and have blood cholesterol and blood pressure levels within the ranges currently recommended. Additional factors are undoubtedly involved. Physical inactivity overweight, obesity, vitamin deficiencies, and many others have been identified or suggested. Of course, the association of a factor with a disease does not prove that the factor causes the disease, nor does it necessarily follow that correcting the factor (particularly at a relatively late age) would prevent the disease.

Unfortunately, it is extremely difficult to undertake experiments which show that a change in lifestyle prevents coronary disease; this would require that many thousands of individuals would agree to be, randomised to change or retain a particular item of their way of life and adhere to this for decades. This is impracticable. We shall, therefore, probably never have proof that a particular lifestyle factor or item of diet is important and those who demand proof before any action are condemning us to wait for ever.Coronary heart disease is not due to a single cause but is the result of the interplay of a number of factors.

Cigarette smoking

The incidence of coronary disease is increased by the consumption of cigarettes. Stopping cigarette smoking reduces the risk of a heart attack although it may take years before ex-smokers reduce their risk of attack to approximately that of non-smokers. One reason for the fall in deaths from coronary disease in the USA and Australia (and to a lesser extent in the UK) is that so many people have given up smoking. A reduction in the prevalence of smoking in the community is probably the most important single step that can be taken to reduce coronary disease in the United Kingdom.

Blood cholesterol levels

1 Population studies have shown that there is a strong relationship between raised blood cholesterol levels and the incidence of coronary disease.

2. When all the trials are reviewed together, it is apparent that a reduction in elevated blood cholesterol levels by diet or drugs leads to a

fall in the number of heart attacks.

3. The reduction of raised blood cholestrol levels can prevent the progression of narrowing of the coronary arteries as has been demonstrated by coronary angiography.

4. The blood cholestrol level is determined both by genetic factors and by diet. In some people, particularly those with familial hypercholesterolaemia, genetic factors predominate but in such people diet and drugs can still play an important role in reducing blood cholestrol levels.

5. People who have a low blood cholestrol throughout their life have a low risk of coronary heart disease. It, therefore, seems sensible for young people to attempt to maintain a low blood cholestrol by eating an appropriate diet throughout life. There is a widely held view that so-called 'healthy' diets are unappelising, but this is not necessarily the case. Coronary disease is relatively uncommon in both France and China.

   Some communities and some individuals appear to be relatively immune to the effect of a high blood cholesterol level. This may be because they have, for some reason, a protection against it. This might be genetic, or it could be that protection is afforded by items in the diet such as foods rich in antioxidants (for example, fruit and vegetables).

6. Some associations have been reported between a relatively low blood cholestrol level and death from a number of cause such as cancer, respiratory disease, and suicide. This is certainly of concern but it has not been shown that the low blood cholestrol level is the cause of these fatal conditions. It is reassuring to note that those countries experiencing a substantial decline in cancer mortality and all cause mortality.

   The association of a low blood cholesterol level with cancer may, at least partly, be accounted for by the fact that cancer (and perhaps precancerous conditions) may directly or indirectly lower the cholestrol level. Dietary habits associated with certain psychological disorders leading to suicide (such as alcholism, depression and anorexia) could also result in a low blood cholestrol level. This topic clearly requires more research.

7. Reducing blood cholestrol level by drugs may not have the same consequences as reducing it by diet because drugs may have specific toxic effects unrelated to their cholesterol lowering action. A great deal more information is still required from randomised controlled studies of new drugs before their effectiveness and safety can be assumed. Until this is available, it is wise to be cautious in the use of cholesterol lowering drugs. The benefits of drug treatment seem to out weight the risks in those

with coronary disease who have high blood cholestrol levels that do not respond to diet.

8. In countries such as the UK, in which most individuals have high cholesterol my international standards, other risk factors such as cigarette smoking, high blood pressure and physical inactivity need to be addressed at least as vigorously as the high cholesterol.

High blood pressure

There is a strong relationship between the level of blood pressure and the risk of coronary disease. Reducting blood pressure reduced the risk of stroke, heart and kidney failure very substantially, but death from coronary disease less so. The reasons for this difference in effect are still unknown, but undoubtedly it is very important that high blood pressure is recognised by periodic measurement in all adults and that its treatment is well supervise.

Physical activity

Physical inactivity is an important independent risk factor for coronary heart disease. Regular physical activity should be encouraged in everyone.

Family history

Some families have a very high incidence of coronary disease at a young age. In most cases, this can be attributed to be inheritance of a gene or genes which carry the risk. In others, it may be due to the fact that members of the same family share the same unhealthy lifestyle. Efforts should be made to identify and screen for risk factors those with a family history of coronary disease at a relatively young age.

Other risk factors

Many other risk factors have been implicated with varying degree of evidence. These include a relatively low birth weight, resistance to the effects of insulin, over weight, stress, clotting factors, and low socio-economic status, etc. Some of these are undoubtedly of importance, but more research is needed to establish their influence in causing coronary heart disease.

Conclusion

There is a substantial fund of knowledge about the causes of coronary heart disease that makes it appropriate to recommend that everyone should stop smoking, that sufficient exercise, and adhere to a diet with a relatively low fat content, especially saturated fat, and a high content of fruit and vegetables. There should be a caution in the prescription of drugs that lower blood cholesterol, but they should be used in individuals who still have high levels in spite of diet if they are known to have coronary disease.

The roles of socio-economic factors, stress, and the newly recognised dietar factors (such as vitamins) require further investigation. More needs to be learned about how risk factors cause coronary disease and how this process can be prevented or reversed. Overall, more research into the cause and prevention of coronary disease is required.

DRUG INTERACTIONS

Dr.M.A.Shah,M.D., Dr.Zaffar Abbas, M.D.** , Deptt. of Pharmacology,*
Govt. Medical College Srinagar.

Polyharmacy is an important factor contributing to interaction between drugs in a patient. These interactions may be grouped as a special class of adverse drug reactions, which are estimated to be 7% of all the adverse reactions due to drugs. Besides 4% of all the deaths due to adverse drug reactions are because of interactions.

1. *Antidiabetic Agents*

It is not unusual for a patient of Diabetos to receive drugs in addition to the antidiabetic therapy. It is in the physcian's interest to understand and consider factors which may alter the tight glycemic control necessary for such patients the addition of other drugs to the therapy being an important consideration.

The drugs whose addition to the oral antidiabetic therapy may result in the alteration of the glycemic control may do so in one of the following ways:-

a) Interactions that augment the hypoglycemic action of oral antidiabetic medication. These drugs include:-

   i) Chloramphenicol, Cotrimaxazole, Flucanozole, Phenylbutazone Sufinpyrazone, Cimetadine:

   These drugs act as enzyme inhibitors, impairing the metabolism of oral antidiabetic drugs leading to increase half life of the hypoglycemic agent, resulting in enhanced effect.

   ii) Allopurinol, Probenacid, Salicylates, Ammonium chloride: decrease the urinary excretion of sulfonylurea drugs or their meatbolites. The first three compete for renal tubular secretion of chlorpropamide. Ammonium chloride is included in many cough mixtures results in prolongation of the half life of chlorpropamide which is pH dependant.

   iii) Chofibrate, Phenybutazone, Salicylates, Sulphonamides: displace sulfonylurea drugs from plasma protein binding sites. The chance of this pharmacokinetic interaction is minimized with the use of Glipizide and Glyburide which bind to plasma proteins exclusively by nonionic interactions in compansion to tolbutamide, acetohexamide, tolazamide and chlorpropamide.

   iv) Insulin, alcohol, beta-adrenergic antagonists, salicylates monamineoxidase inhibitors, guanethidine have intrinsic hypoglycemic activity.

b) Interactions that shorten or decrease the effect of the hypoglycemic effect of oral antidiabetic medication. These drugs include:-

   i) Rifampin, Phenytoin, Phenobarbitone, Carbamezapine, Chronic access of alcohol. These drugs are potent enzyme inducers resulting in rapid metabolism clearance leading to reduced plasma concentration of the drug and reduce hypoglyeemic effect.

   ii) Corticosteroids, Dexltrothyroxine, Epinephrine, Oral contraceptives, Acetazolamide, Epinephrine, Thiazides have intrinsic hyperglycemic activity.

Drugs which alter the effect of Insulin/ Sulfonylureas

a) Causing agumentation of the hypoglycemic effect of Insulin:

i)	Alcohol	Impaired gluconeogenesis and increased insulin secretioncontribute to hypoglycemia.
ii)	Beta Blockers	Inhibit glycogenolysis and unmask the sympathetically-mediated symptoms of hypoglycemia.
iii)	Oxytetracycline	has been shown to enhance the hypoglycemic effect of insulin. Mechanism not known.
iv)	ACE Inhibitors	Improve insulin sensitivity. Reduce gluconeogenesis possibly by increased bradykinin level.

b) Drugs reducing the hypoglycemic effect of Insulin/Sulfonylureas

i)	Calcium Channel Blokers Diazoxide Phenytoin Clonidine	all these drugs inhibit insulin secretion directly.
ii)	Glucocorticoids Sympathomimetics and other drugs as in(b. ii).	have intrinisic hypoglycemic activity. Glucocorticoids also inhibit insulin secretiion directly. Diuretics do so by indirect mechanism i.e.by depletion of K+.

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Letters to the Editor

King Faisal Specialist Hospital
And Research Centre
Riyadh 11211

My Dear Dr.Malik,

Everyone here is appreciative of your dedication and efforts. From here too, people are very serious about contributing to this project of yours and I am at it my self. The more I learnt about the current status of health care in our state the more convinced I was to admit that SMHS Hospital is atleast functional. I said this to you there only.

I shall keep in touch and please be totally assured of my support for the much needed Medical periodical you are starting. May you continue to flourish!

Yours sincerely,

Sd/-
Ashraf Jallu
M.S.FRCS
(Neurosurgeon)

Dated:1.7.1994

Dr. Anil Arora
Consultant Gastroenterolosist
Sir Ganga Ram Hospital,
New Delhi-60

15.7.1994

My Dear Dr.Malik,

I am pleased to know that you have been actively involved in publishing the journal called 'JK Practitioner'. I wish you all the success.

From my side I shall be contributing to the journal at the earliest.

Yours sincerely,

Sd/-
Dr. Anil Arora

Buffalo, New York

Sisters Hospital
Answering the call

Khalid J, Qazi, Md, FACP
Associated Chief of Medicine

I am delighted to know from your recent letter that you have taken an initiative to produce a quality medical journal, JK Practitioner, from our alma mater, Medical College of Srinagar, Kashmir. It is a huge task and I commend you for taking this initiative which was long overdue. I would certainly encourage all of our friends here in the United States to patronize this endeavour to the best of their abilities and would do so myself, as well.

Congratulations and good luck for this initiative

Sincerely,

Sd/-
Khalid J.Qazi,
Associate Chief of Medicine

Cardio-Thoracic Centre
All India Institute of
Medical Sciences

Upendra Kaul
MD, DM, MNAMS
Professor, Cardiology
New Delhi-110029
(India)

Dear Dr.Malik,

It will be my pleasure to be associated with JK-Practitioner (Journal of Medicine). You can put my name in the advisory board or editorial committee etc. I will contribute to the journal, do let me know the format etc.

With best wishes.

Yours sincerely,

Sd/-
(Dr. Upendra Kaul)

University Hospital of Wales

Department of Medicine

Dear Dr.Malik

I am delighted that you are starting a Journal and I wish you every success with it. It is very difficult to break into the big league even for those Journals which start in the West, but if you continue with hard work and publishing good material there is some chance of success. I will try to put something together for a review article and as soon as it is finished I will send it to you.

Kind regards,

Yours sincerely,
Sd/-
Dr.M.A.Mir, MRCP.
Senior Lecturer and consultant

Dr.G.R.Nahami
Best of Health Clinic
I Andover ST.
Sheffield
S3 9ED

Dear Editor,

First let me congratulate you on publishing a medical journal from Srinagar. It is exciting news and I wish you all the success in this enterprise. This is a wonderful undertaking and will be beneficial for the medical profession in particular and public in general.

I shall be more than delighted to contribute to your journal in future. I shall be particularly interested in subjects like dermatology, diabetes, backache and migraine. Please let me know your preferences for the next publication .

Once again I wish you all the success in launching of this medical journal.

With regards.

Yours sincerely,

Sd/-
Dr.Ghulam Rasool Nahami
M.B; B.S. D.P.D.(Dermatology)
General Practitioner Principal.

Head Department of Cardiology
Postgraduate Institute of Medical
Education and Research,
Chandigarh-160012, (India)

Dr.R.P.Sapru
M.D., Ph.D(Edin) F.A.M.S., F.I.M.S.A., F.I.C.A.

Dear Dr.Malik,

I am delighted that you are starting a new journal for medical practitioner. I am sure, it will be a great success. I would be very happy to be of any assistance in this regard.

Best wishes,

Yours sincerely,

Sd/-
(R.P.Sapru)

Nassau Country Medical Center
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Faroque A Khan., M.B., F.A.C.P., F.R.C.P(C).F.C.C.P
Chairman, Department of Medicine

Dear Dr.Malik,

I am sure your experience, training and dedication will be greatly appreciated by the people of Kashmir.

Keep me informed and I will try to help as best as I can.

Best wishes to you and your family.

Yours sincerely,

Sd/-
Faroque A.Khan.M.D

King's College School of Medicine and Dentistry
Department of Haematological Medicine (U.K)

My dear Malik,

I should like to congratulate you on the initiative, which is a creditable idea, and I assure you that I will do everything possible to make it a success. As you know, whatever the melieu, or the surrounds of an individual, the roots, the past and the joyous youth can and never should be forgotten, for it ensures sanity and to a certain extent cultural continuity.

As you know I am on the editorial board of a number of journals and have first had experience of everything that is involved. However, JK Practitioner is a different story, come what may I shall contribute and serve in any capacity you want me.

We could and should try and get a truly International Editorial Board, so that the Journal gets into the Index Medicos.

I wish the journal all the success.

Yours sincerely,

Sd/-
G.J.Mufti.
Senior Lecturer/Hon Consultant
MBBS, DM, FRCpath, DTM & H.

Date:1.8.1994

Dr.Mir Mohd Ashraf
MRCP, FICA, FACCP (USA)
Physicain Specialist Cardeologist, Cresclent I.C.C.U, Boulevard Srinagar.

Dear Dr.Malik,

I am sure you are fine, It is a good idea and have a journal of our own state. In future I would like to write more articles.

I would like to congratulate you, your advisors and editorial committee.

With regards.

Yours sincerely,

Sd/-
Dr.M.M.Ashraf.

"CHOIEDOCHOSCOPY-AN ESSENTIAL INTRAOPERATIVE PROCEDURE"

Prof.N.A.Wani, (M.S, FRCS, FICS), Head Deptt.Surgery SKIMS Soura, Sgr.Kashmir (India).

Diseases of the biliary tract are the commonly encountered problems in the surgical practice, and their management has been a challenging task for the surgeons throughout the world. The peroperative diagnosis of the calculous diseases of the biliary tract poses no problem, however the duct stones are so often missed (2 to 28% Forgarty et al 1968) that they are a disturbing postoperative complication for both the patient and the surgeon. Blame is attached, however, to the efficiency of the instrumental exploration of the ducts and above all the reliability of the means of checking them, and verifying the complete clearance of common bile duct, once all stones are considered to have been removed. The fundamental defect of this means of exploration and removal of stones is that the maneuvers are made blindly and therefore cannot assure complete removal of all stones (Burkett et al 1981).

The use fullness of the cholangiography used as a check after instrumental bile duct exploration is clearly shown by the study of Legrand and Cave (1978), who obtained insufficient or incorrect results in 65% of the intraoperative examinations. According to Hall (1973); the use of completion cholangiography via a T-tube has not decreased the incidence of post operative residual stones.

Residual or retained stones after common duct exploration are a persistent problem in the management of biliary calculi. Despite enthusiasm for both the instrumental extraction through the T-tube fistula and other non operative dissolution techniques, most such stones require removal at a second operation. The incidence of the retained stones has been reported to be 7 to 20%. This represents a rather high occurrence considering that many of these patients require reoperation for removal of the retained stones. Although the operative cholangiography has been credited with reducing the incidence of the retained stones, the incidence still remains about 7% in recent series and no matter how carefully the operative cholangiography is performed the false positive and false negative error rate remains 4 to 14% (Cooperman et al 1982), indicating a questionable role for operative cholangiography.

To overcome these difficulties in the management of the biliary tract disease various methods for operative examination of the biliary tract have been in use, and of the various modalities available, choledochoscopy is now being increasingly used alone or in conjunction with other modalities to improve the overall diagnostic and curative rate. With increasing improvement in the fiberoptic technology and technique of procedure and distinct advantage of direct visualisation, the choledo-choscopy is becoming a safe and reliable procedure and is new being advocated in every case in which there is an indication for the exploration of the common duct, especially in the calculus disease of the biliary system. The biliary endoscopy has become more reliable procedure than the peroperative cholangiography as it has reduced the incidence of the retained stones to 2 to 3% (Motson et al 1990).

Choledochoscopy is quicker and readily repeatable than peroperative cholangiography, and adds only a few minutes to the operative procedure. It is also unaffected by the air bubbles entering the duct system and avoids the unnecessary radiation exposure to the patient and the staff performing the procedure. Biopsy can also be taken of any suspicious lesion through the choledochoscope. It is the only method to diagnose cholangitis in vivo. The sphincter can also be seen in action, and it has also proved helpful in the management of hepatic duct stones.

Use of the choledochoscopy is also advocated, in addition to the conventional criteria, for assessing resectibility in all patients with ductal or periampullary carcinoma. Needless radical procedures can be avoided by routine use of choledochoscopy, where indicated, and the long term cure rate after adequate resection will be tremendously improved by identifying those patients with truly localised lesion. Choledochoscopy can also be used post operatively through a T-tube tract for extraction of any residual stones.

Apart from detecting the retained stones in the biliary tract, choledochoscopy is also helpful in diagnosing other biliary tract pathologies like:

- Congenital diaphragm
- Inflammatory lesions
- Diverticula
- Strictures
- Foreign bodies
- Extrinsic compression
- Neoplasms

Moreover, choledochoscopy is a safe and simple procedure and in itself is not associated with any complication and has gained a wide acceptance for its use, though not universally accepted. A survey reported by Puglionisi (1978) at the 80th Congress of the Italian Society of Surgery stating that choledochoscopy was used by 68.2% of the British surgeon, 57.6% of French surgeons and 14.8% of the Italian surgeons.

Schein and Hurwitt (1962) described the usefulness of choledochoscopy for evaluating the interior of biliary tract at the time of operation especially to see the sphincter in action, to look into the ampulla and to recognize cholangitis. However, the various advantages and limitations of choledochoscopy were also described.

Advantages	Limitations
1. Direct control by surgeons	1. The technique has to be learned
2. No contrast medium needed	2. Choledochotomy is required
3. Direct differential diagnosis.	3. Pancreatic duct is not visualized
4. Sphincter can be seen in action	4. Duct may be too small for visualized.
5. Immediate availability	
6 No auxiliary person or equipment required.	
7. Unlimited number of examination	
8. Live anatomy.	
9. Only way to diagnose cholangitis in vivo.	

Schein (1969) subjected patients to peroperative choledochoscopy and intraoperative T-tube cholangiography, and made the following observations:-

i. Choledochoscopy was fund to be safe and uncomplicated procedure for the evaluation of the dilated CBD encountered in cholelithiasis.

ii. Choledochoscopy was helpful in differentiating calculi and air bubbles in the contrast media of cholangiograms and obviated the need for repetitive explorations and cholangiographic studies.

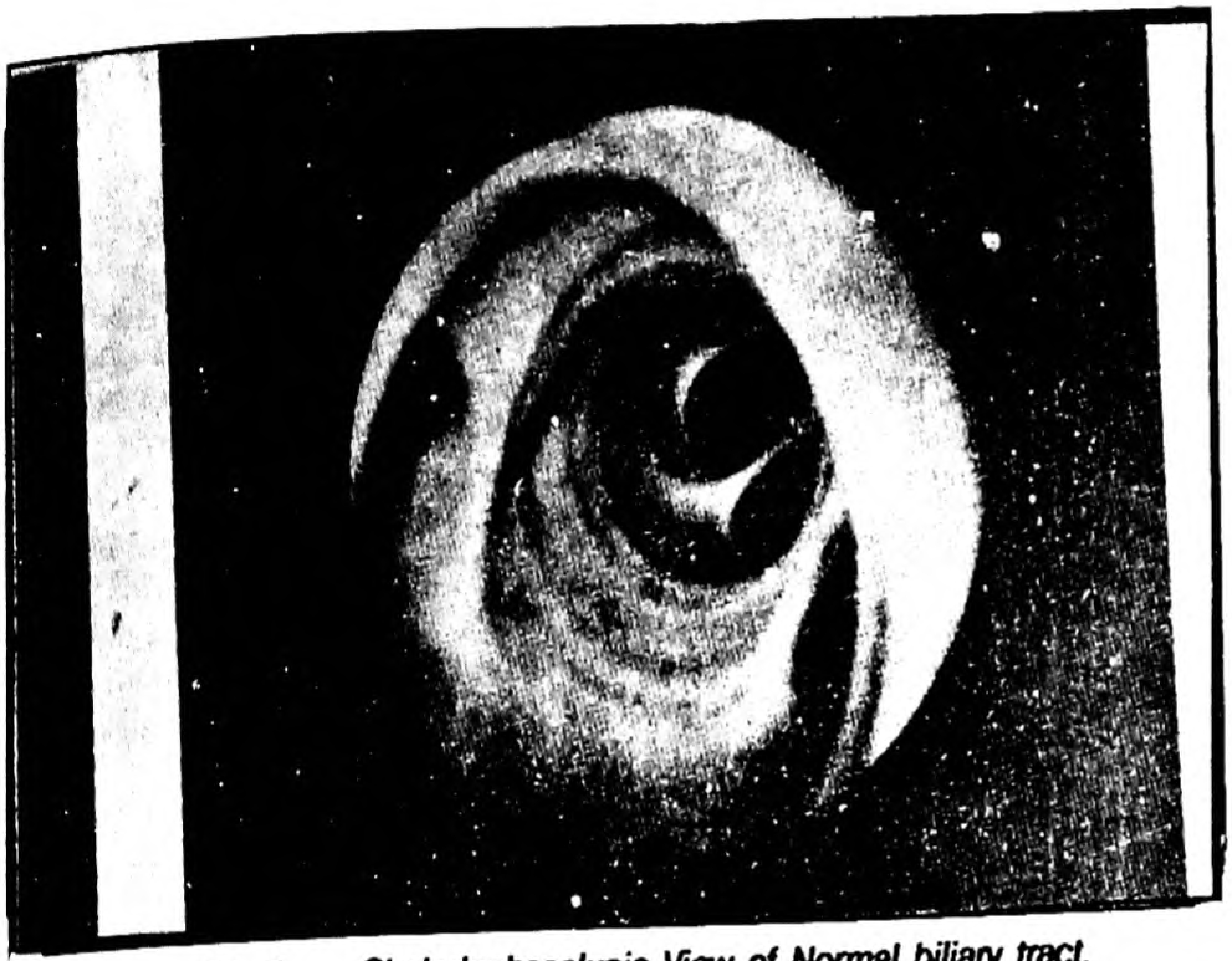
iii. In 117 examinations an assumed retained calculi rate of 5.1% observed with standard instrumental exploration was reduced to 0.9% when endoscopy was performed.

iv. The combination of choledochoscopy and the operative cholangiography avoided the post-operative residual stone problem.

False negative results with the use of choledochoscope range from 0.4% (Motson et al 1980). This depends on the experience of the examiner besides other reasons like failure to seen the biliary tract systematically and completely including the LHD and failure to see the lower segment of CBD and ampullary orifice.

Development of the choledochoscope:

The first reported common duct exploration dates back to 1887, when Thronton described two successful cases in which CBD stones were crushed and removed via the dilated cystic duct. He later described two other cases in which the stones were removed through a choledochotomy and the lower end of the duct was inspected with a glass speculum. In 1890 Courvoisier described a case in which he reoperated to retrieve a retained stone. The earliest attempts at the peroperative visualization of the biliary tract was made by Bakes in 1923. He described a speculum with a proximal mirror which used reflected light from the surgeons head lamp. It was not until 1941 that the first rigid optical choledochoscope was developed by Mciver. It was a modified cystoscope, 5mm in diameter with a right angle 7cm from the tip. IT provided an irrigation channel and a light source. In



(Fig.I) Choledochesclupie View of Normal biliary tract.

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1953 Wildeglan of Germany improved the choledochoscope by incorporating an optical system with a light source and an irrigation channel incorporated in a rubber sheath. By 1970 the Hopkins rodlens optical system had been developed which was incorporated in the Karl Storz rigid choledochoscope and offered a wide angle of view with minimal distortion of the image.

In 1965 Shore and Lippman described the first flexible. This was a 50cm long with an external diameter of 6mm. It contained an optical lighting system and an irrigation system housed within a single latex andvinylcopolymer plastic sheeth. The optical system considered of a flexible fiber-optic bundle 2.5mm in a diameter composing of 36000 coated optical glass fibers each. 0004 inch in diameter. These fibers were so aligned that they would convey a clear image from the distal forward viewing objective lens to the proximal eyepiece through a variably curved pathway. This flexible choledochoscope was found to be easier and safer to handle than the rigid counterpart. In 1971 Shore and Morgenstem designed an improved miniaturised right angle choledochoscope based on the Hopkin optical system. This optical system permitted the miniaturisation so that the 5cm long exploring horizontal limb incorporating optical lighting and irrigation system measured only 5x3mm in cross section. The right angked construction and compact design eliminated the difficulties encountered with bulkier Wolfe and Wildegans scopes and also the manipulative problems associated with flexible fiberoptic instruments were overcome. The wide viewing angle, extreme depth of field and fixed focus provide the exquisite image quality so essential for prompt endoscopic orientation.

The CHF series of choledochoscope was introduced by Olympus with improved fiberoptics and maneuverability. These flexible choledochoscopes were gradually improved and modified by Ashby to the present range, the CHF-20 series. In 1987 the choledochoscope was further modified by Morris to an ultra thin model with an external diameter of 2.5mm and a channel diameter of 0.5mm. Currently choledochoscopes of 0.8-2mm diameter with optical illumination channel are being used to visualize the biliary tree. These can be introduced percutaneously, via the duodenoscope working channel or via the cystic duct.

Method :

After exposure of the CBD by any of the standard incisions, the choledochoscope is introduced through a supra duodenal choledochotomy incision. At inferiorly placed choledochootomy incision is preferred as it may be converted into a choledochoduodenostomy if a drainage procedure is subsequently found necessary. Routine instrumentation of the ducts is carried out with standard instruments like Desjardin's ferceps and balloon or irrigation catheter. The choledochoscope is then used to detect and remove any stones not removed by the conventional exploration.

The examination is carried out in a fluid medium of the sterile saline infusion, at the room temperature through the irrigation channel. The rate of infusion should not be at a high pressure as it may force the bacteria into the sinusoids, risking infection especially in the patient with biliary infection.

Systemic exploration is essential. Proximally good views can be obtained of the right and left CHD and dependingupon the choledochoscope used, two or three main divisions of the intrahepatic ducts. (Fig.I) This is the easier part as the bile ducts are supported by the liver tissue. After examinations of all the ducts proximally the choledochoscope is withdrawn and reinserted into the CBD. At this point the rate of flew of the saline should be adequate because the ducts may collapse. The CBD is inspected distally upto the papilla of water. It has get a resseted appearance endoscopically. If there is a doubt regarding the visualisation of the papilla, a fogarty catheter should be passed under direct vision through papilla into the duodenum and balloon should be inflated and the 5cm gradation mark on the catheter identified. The catheter is then showly withdrawn until the

1cm mark is reached. At this point the papilla can be viewed.

If the stones are found, they may be grasped with the forceps, retrieved with dormia basket or with the aid of a balloon catheter. If the stone is impacted, it may be crushed with reinforced basket forceps and then retrieved.

When the choledochoscopic examination is complete, the ducts should be closed with absorable suture and with T-tube drainage.

A T-tube cholangiogram should be taken on the 7-10'th post operative day to exclude retained stones and to confirm the good drainage of bile into the duodenum.

The conclude, it can be said that the persperative choledochoscopy is an adjuvant to the standard CBD exploration is a useful addition to the armamentorium of the biliary sugeons. It no doubt increases the decreased if not completely eradicated the problem of the retained CBD stones. It is strongly recommend in every case where exploration of the CBD is indicated.

([illegible])

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"Unusual Presentation of Gastric Lymphoma"

*Gazanfar Ali, **M.A.Kamili, *Samia Rashid, ***G.H.Reshi, ***G.M.Malik, *****G.Q.Allaqaband.
M.D M.D M.D M.D M.D FRCP

Introduction

The gastro-intestinal (GI) tract is the most common extranodal site of non-Hodgkin's lymophoma[1]. Primary gastric lymphoma form 2% of all lymphomas[2]. We report in case of gastric lymphoma with an unusual presentation.

Case report

A 50 year old obese female presented with fever of 6 weeks duration without any other associated symptoms related to respiratory, cardiovascular, genitourinary or gastrointestinal system. Before reporting to hospital, she had attended different medical practitioners and received different antibiotics, despite the fact that various investigations were inconclusive. Examination revealed anaemia and fever. She did not have any lymphadenopathy, chest signs or hepatosplenomegaly other system examination was normal. Haemogram revealed Iron deficiency anaemia (Hb 9gm%). Blood chemistry, liver function tests and renal parameters were normal. Blood culture was sterile. Serology for enteric fever, brucellosis and rheumatoid arthritis were negative. ANA and L.E. cells were negative. X-ray chest P.A.view, abdominal ultra-sonography, Kub and I.V.P were normal. Bone narrow examination showed evidence of Iron-deficiency with no abnormal cells or acid fast bacilli. She continued to have high grade fever. She was empirically put on antitubercular treatment without any significant improvement. By now patient had developed sptenomegaly. Barium X-ray stomach showed irregularity of greater curvature (Fig.I). Endoscopy revealed a nodular ulcerated lesion in the body of stomach both along greater and lesser curvature with narrowing of lumen Multiple biopsies taken, showed non-Hodgkins lymphoma (low grade). C.T.scan of abdomen revealed a thick walled stomach without any irregularity, with splenomegaly. There were no lymph nodes(Fig II). Pt was given combination chemotherapy. She refused to undergo total gastrectomy. After 2 doses of CHOP therapy, repeat GI endoscopy revealed 90% regression of the lesion. She was given third course of chemotherapy but 2 weeks later was admitted with high grade fever, dyspnoea.

On examination she was febrile, tachypnoec, toxic, cyanosed with signs of bilateral consolidation. Abdominal examination showed a hard nodular, globular fixed mass 6'x5" in the epigastric region. She was treated for severe sepsis but died on second day of admission.

Discussion

Primary malignant non-Hodgkin's lymphoma of the GI tract are extranodal lymphomas that originate form mucosal lymphocytes. They are usually of low grade malignancy[3]. The primary gastric lymphomas from 7% of all gastric malignancies and 2% of all lymphomas[2] hence, relatively uncommon. They present with early satiety, epigastric pain and fatigue, while this case had no GI tract symptoms but only fever. A clinical diagnosis is much less frequent in such cases. Systemic complaints are very uncommon in primary gastric lymphomas[4] as also in non-Hodgkins lymphoma[5]. The present case signifies the importance of upper G.I. endoscopy in patients with prolonged fever. Non-Holdgkins lymphona of G.I.tract differ from node based lymphomas in that they may be curable by radical resection, yielding a 10 year survival rate of 46% in gastric lesions[3]. One in 4 patients with non-Hodgkin's lymphoma will have involvement of G.I.tract and may benefit from upper G.I.endoscopy-a fact worth knowing if it applies to patients worldwide with this type of lymphoma[6].

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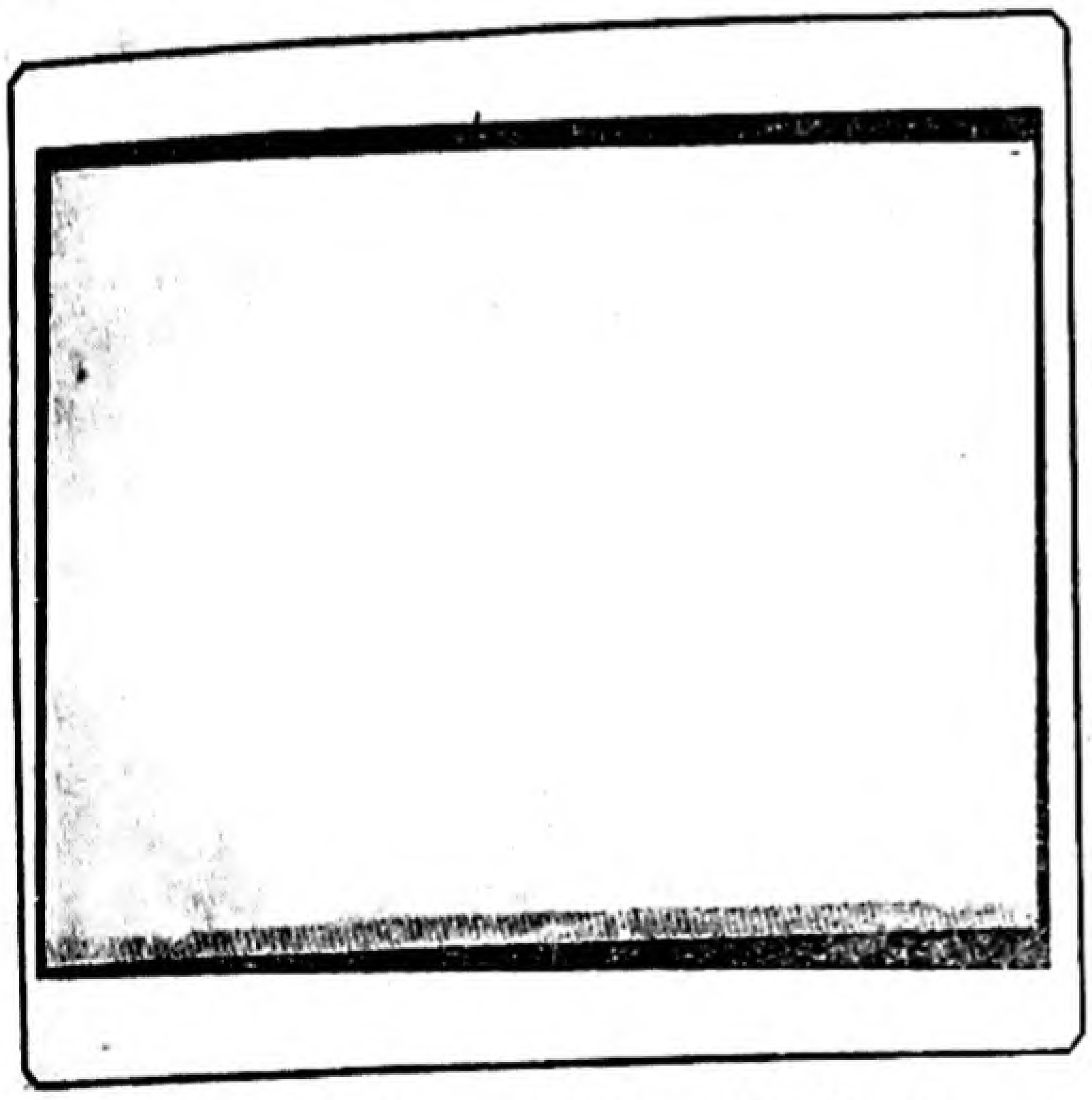

Fig-I: *Ba meal X-ray of stomach shows irregularity along greater curve.*

Fig-II *CT scan of stomach (with contrast) shows marked thickening stomach wall with irregularity of mucosa.*

A Case of Jaundice

G.M.Malik, M.Mubark, S.Kadala, T.Hussain, GIT Division, G.M.C.Srinagar.

Case report:

A 60 year old male was admitted with 15 days history of yellowish discolouration of both eyes of insidious onset and rapid progression. He also complained of anorexia, generalized weakness, pruritis and passing clay coloured stools one examination, he looked anemic but of on large built and had itch marks all over the body. Abdominal examination revealed a firm palpable liver 3cms below the right costal margin with smooth surface and rounded margins. Rest of the systemic examination was normal.

On investigation, haemoglobin was 9gms/100ml. ESR was 31mm/Ist hour, (Wintrobes method was) stool was clay coloured and positive for occult blood. Total serum bilirubin level was 20mg/100ml and alkaline phosphates 80 KA units/litre. Rest of the haemogram and the Blood chemistry, ECG, X-ray chest (PA view) was normal.

USG revealed an enlarged liver with normal echogenicity without any evidence of secondaries, but the intra-hepatic biliary ducts were dilated. Gall bladder (GB) was not visualized. There were two hyperechoic shadows at Porta-hepatis without acousting shadowing (Fig.1). Cholangiocarcinoma with secondaries in Porta-hepatis was kept as one of the possibilities.

CT scan abdomen revealed enlarged liver with dilated intrahepatic biliary ducts. The main hepatic ducts, especially the left one, were dilated. A few hypodense areas with abnormal enhanced, especially in the left lobe and two hyperdense areas, at the porta-hepatis were noticed. Gall bladder was not visualized (Fig.II). This confirmed the diagnostic possibility of USG. CRCP, done one week later, showed narrowing in the upper part of CBD and post contrast films revealed dilated intrahepatic biliary ducts. The diagnosis of growth (cholangio-carcinoma) in the upper part of CBD was documented. The growth being inoperable, a palliative ERCP stent was negotiated at the same time. The patient improved clinically and serum bilirubin and alkaline phosphates levels also came down. But, ultimately the patient deteriorated and died about one month after stenting, probably because of the ascending cholangitis and subsequent septicemia.

Discussion:

Patients with cholangiocarcinoma usually present with painless jaundice, prutitis, weight loss, and acholic stools. Jaundice may be delayed if only one main duct is involved. The patient may have either an epigastric or deep seated vaguely localized right upper quadrant pain. Fever is present when associated with ascending cholangitis. Because of the gradual obstructing process, cholangiocarcinoma is often far-advanced by the time of diagnosis. Hepatomegaly is a frequent finding on examination[1,2,4],. Our patient had involvement of main duct in its upper part, with the result the clinical appearance of jaundice was delayed and by the time the diagnosis was made, tumour had already spread.

Liver function tests reveal a cholestatic jaundice with very high bilirubin levels. The feaces are pale and faty and occult blood is often present[1]. USG is particularly helpful, showing dilated intraheptic bile ducts. It can sometimes identify the tumour mass or even secondaries[3,4].

C.T. scan shows intraheptic biliary dilatation, but the tumour is different to demonstrate as it is isodense with rest of the liver[3,6]. However in our patient, C.T. diagnosis was more specific, showing some hypodense areas in the left lobe and Porta-hepatis in addition to intra-hepatic biliary dilatation.

Endoscopic or Percutaneous cholangeography or both is essential and should be performed in all patients with the picture of cholestasis and dilated intrahepatic ducts, shown by USG or C.T. ERCP clearly demonstrates obstruction and post contrast films reveal dilated intrahepatic ducts[2,3,5].

The resectability of tumour is only 20%. Tumour of the lower bile ducts may be resected with a one year survival rate of 60%. More distal tumour need extensive surgery or are inoperable. The inoperable group is subjected to palliative stenting procedure which may be combined with chemoradiotherapy[2,3,6]. The tumour is ultimately fatal. It kills by its site, making it inoperable, rather by it malignancy[4].

Our patient had an inoperable growth that was subjected to pallivative ERCP stenting, but he died one month after procedure.

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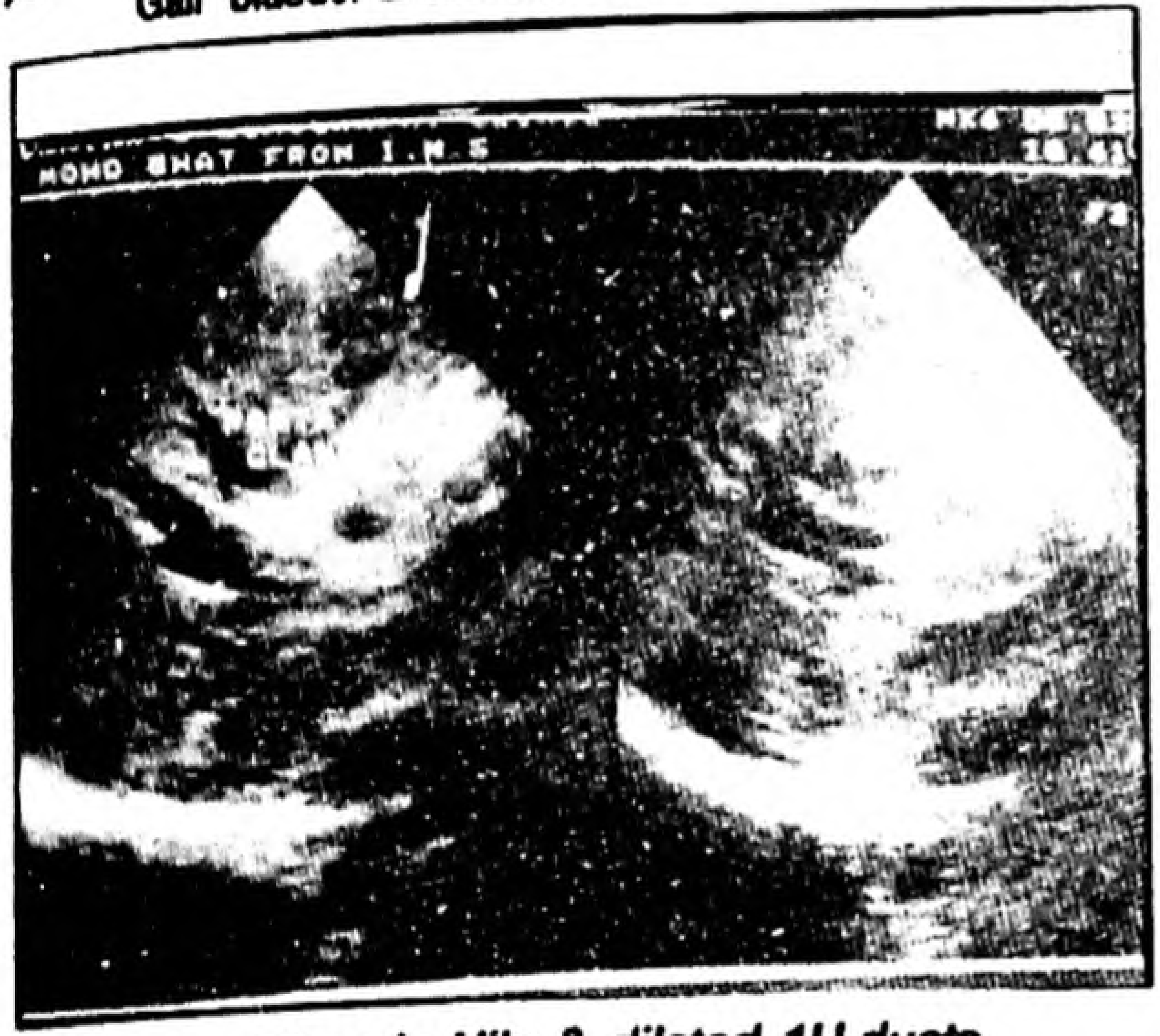
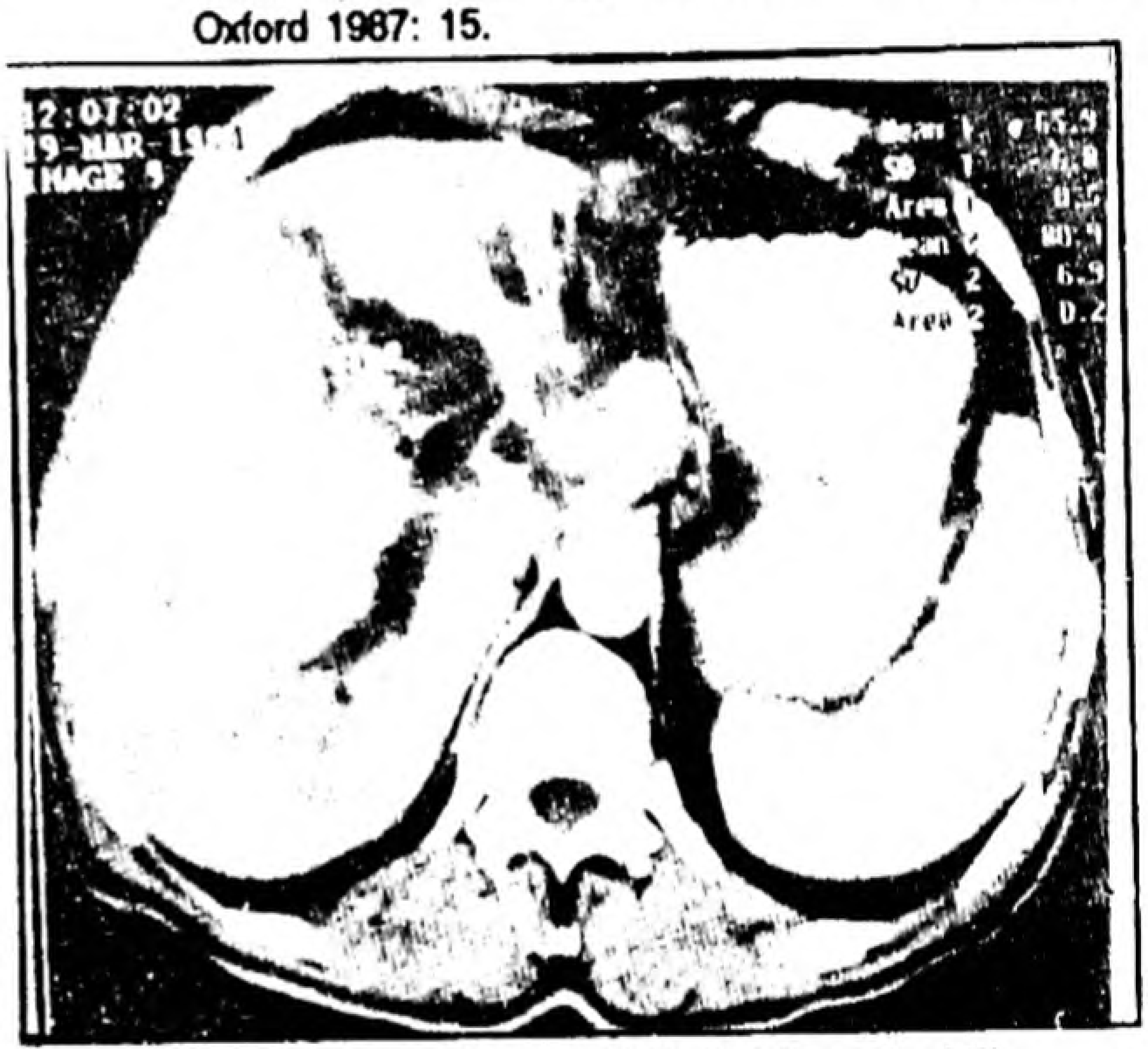
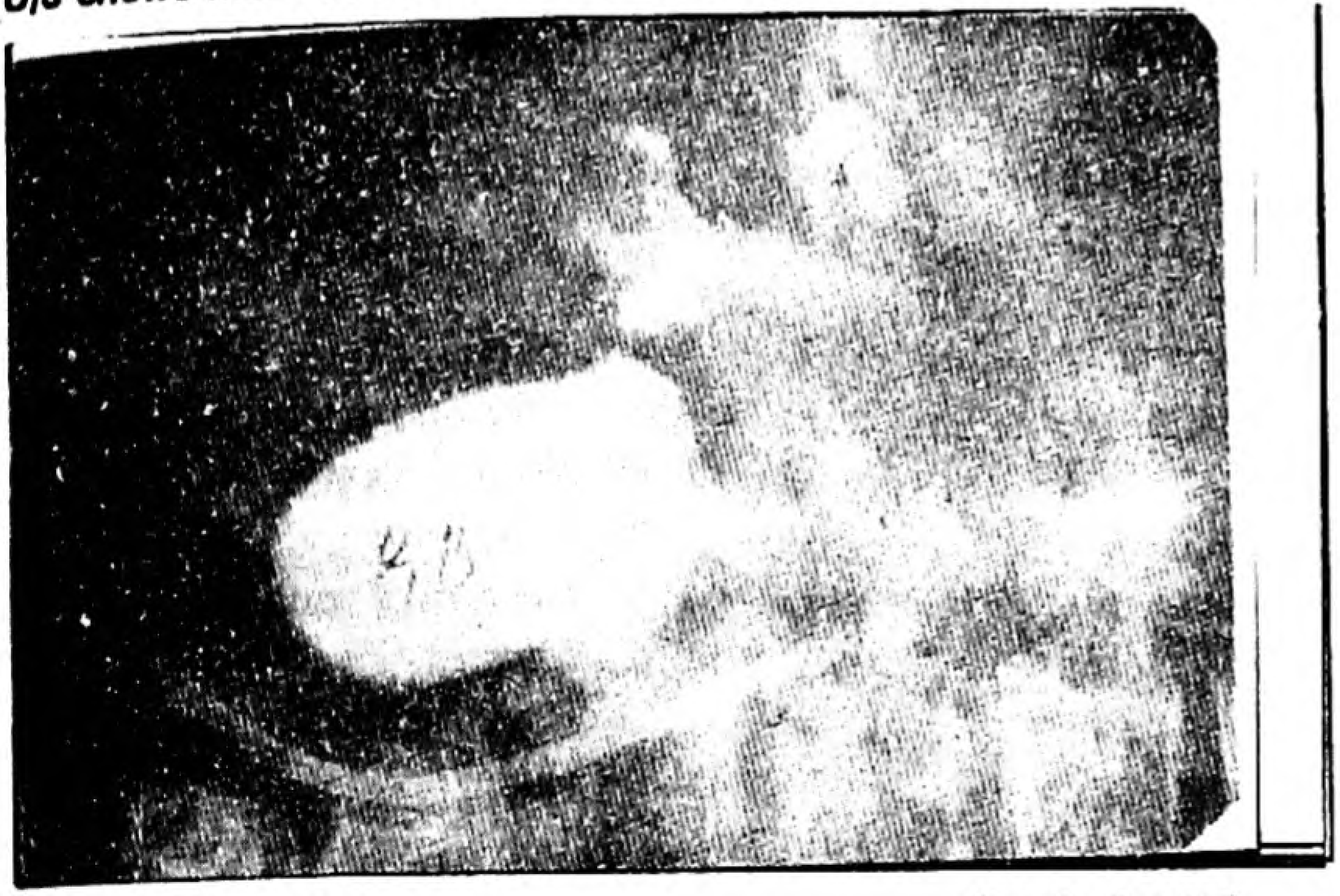


U/s shows Mass in Hila & dilated 1H.ducts



CT scan shows dilated H.D & Mass in Hila



ERCP, Stent at Stricture

(31 A, 9-4-94)

Bone Marrow Aspiration

Dr.M.L.Bhat[*], Dr.Mansoor Laherwal[**]

Bone marrow aspiration is the most important means of assessing bone marrow function and malfunction and is second only to examination of Peripheral blood as a screening and search procedure of disorders directly or indirectly affecting the marrow.

Normally there are about six hundred grams of haemopoietic marrow in the skeleton of an adult individual of which 70% is located in the pelvis, vertebrae and sternum. The usual sites for bone marrow aspiration in adults are the iliac crests and the sternum, while as the sites for infants are antero-medial aspect of the tibia which is quite safe and convenient. In older children the spinous processes of upper lumbar (L1-L8) and lower dorsal (D10-D12) remain richly cellular and can be used as the sites for aspiration as these can withstand the firm pressure applied during the procedure.

Procedure

The needles used are "Saleh" and "Klimia". Under sterile conditions and the patient lying supine, an overlying area of 1 inch being the superior iliac spine is infiltrated with 1% Xylocain and the periosteum also. The needle with its obturator fixed in place and guard adjusted is pushed through the skin and is inserted forcefully in a horizontal direction by twisting it back and forth till the cortex is penetrated. The penetration is sensed by a sudden "Give" and the operator notes that the needle feels planted firmly. The stylet should be withdrawn and within 5 to 10 seconds thick dark red marrow normally Oozes. With the help of a 10ml syringe fitted to needle 0.2 to 0.5ml marrow is aspirated and smears made on clean glass slides after allowing blood to run off leaving behind pale particles. The stomal particles can then be air dried or immersed in a fixative for histological examination. The needle is then withdrawn and firm pressure applied for 5-10 minuted.

Certain haematologists prefer sternal aspiration because of the advantages over the superior iliac spine such as less density of the bony cortex of the sternum and in addition being flat, superficial and stable.

Indications

A) **Absolute:**

1. Megalloblastic macrocytic anaemia.
2. Aleukemic or sublejukemicleukemia.

B) **Diagnostic importance:**

1. Multiple myeloma.
2. Aplastic anaemia.
3. Gaucher's disease.
4. Storage disorders.

C) **Confirmatory importance**

1. Leukomias of all types.
2. Haemolytic anaemias.
3. Idiopathic thrombocytopenic purpura.
4. Idiopathic granulocytopenia.
5. Parasitic infectations (Leishania and Malarial parasite).
6. Metastatic carcinomas.
7. Mydoproliferative syndromes.
8. Iron deficiency anaemias.
9. Lymphomas (staging).

Contraindications

Absolute:

1. Haemophilia.
2. Bleeding diathesis.
3. Infections.

Complications

General:

1. Introduction of infections such as abscesses and osteomelitis of bone.
2. If aspiration site is sternum, complication are as under:
   - a) Rupture of aorta.
   - b) Mediastinitis.
   - c) Pneumothorax.
   - d) Haemothorax.

Medical Education Need for Reorganization

Prof.M.Y.Bhat, Deptt.Medicine, Medical College Srinagar(Kashmir).

Aims and Objective of Ideal Medical School

The main aim of medical education is to produce an educated doctor will versed with the principle and method. The primary aim of the medical school should not only depend upon imparting only a fixed quantum of knowledge through text books and lectures and an examination system based on retention and recall as by this approach the whole system of Medical education is going to come under strain as the medical education is growing at a fast race. The primary aim of medical school should be to consider spirit and priority which should revolve round "add to the knowledge policy" The medical education should foster essential qualities among physicians. He should be trained as a

- Scientist
- Humanist
- Skilled examiner and doctor.
- And a Judge (Medical)

Medical schools need detailed and constant reorganization and apprisal for purpose of planning a particular training programme. For appraisal of Medical Education following factors are involved:-

1. Student
2. Teacher
3. Course and curriculum
4. Assessment and test

Student

A well developed selection need to evolve to select students for medical education. This should among other factors include:-

a) A pre-medical professional knowledge
b) Attitude
c) Value structure
d) Intellectual level
e) Character (Physical and moral development)
f) Sense of service and discipline
g) Aptitude
h) Missionary spirit

Medical schools should aim at efficient delivery of Medical Education which should be more practical and problem oriented. A Medical School should have,

a) A requisite student teacher proportion
b) Well developed laboratory backup.
c) Efficient and developed hospital service need to present to make a better medical student. Besides modern methods of computerised Medical record system and also a local Medical School Journal to highlight local experience and research.

Postgraduate Education

The post-graduate medical education need special and constant review with a particular objective of obtaining high academic standard and planning.

This academic objective is a pre-requisites for production of :

a) Efficient teachers
b) Research workers
c) Specialists and
d) Leaders and policy makers of academic progress.

Teacher

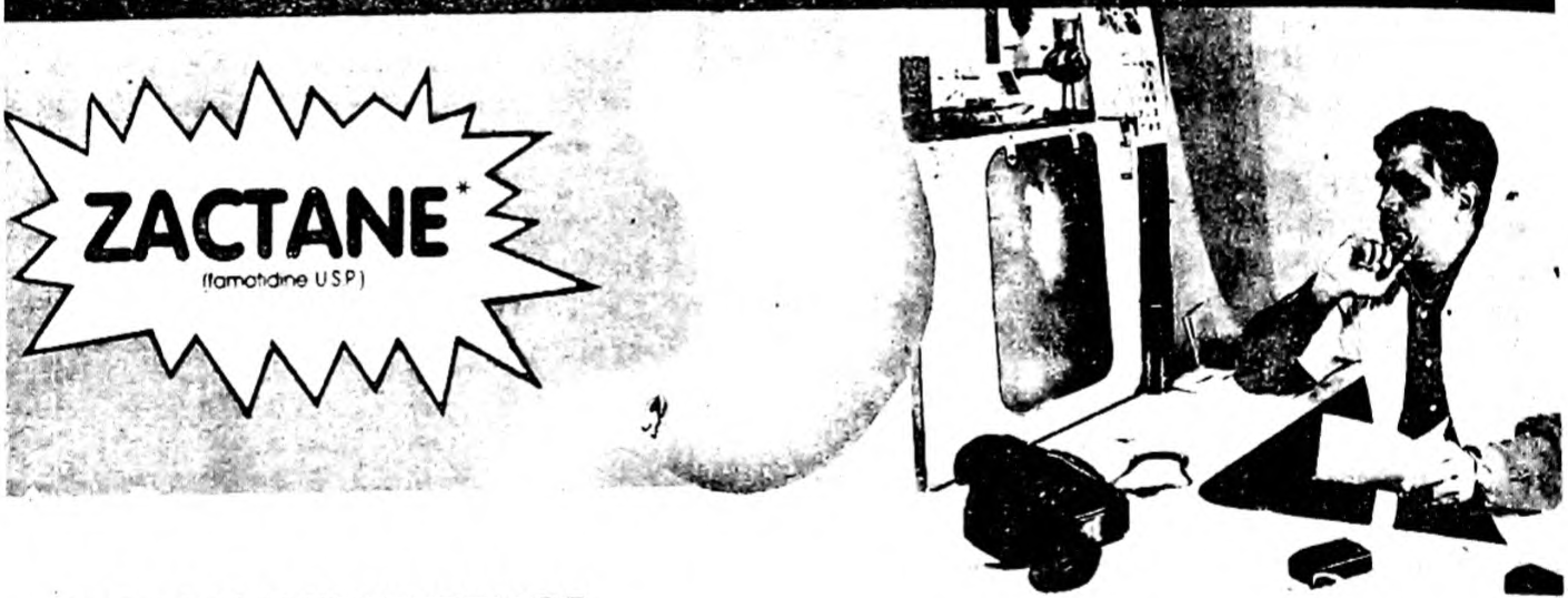
Selection of Medical Teachers should be

made more objective and appitude for teaching,

- Goodness
- Dedication
- Zest for updating knowledge
- Receptiveness for learning process
- Honesty
- Regularity
- Integrity

Attention need be paved to :

1. Better teaching ability and adequately rewarding
2. Performance indicator of education competence be identified by a feed back from students
3. Medical teachers oriented to teach in full instructional methods.
4. A medical school level teacher be encouraged
   a) To take part in symposia,seminars
   b) Interaction with other colleagues and Departments.
   - Refresher courses
   - Fellowship awards
   - Promotion and renumeration awarded on clinical/teaching ability.
   - Encouraging a full time teacher supplemented or complimented by part time staff.



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Highlights of Symposium on Tuberculosis Held at Chest disease Hospital in Sept.1994

10.A.M.	Welcome Address:	-	**Dr.Abdul Quyoom** Medical Suptd. C.D.Hospital
10.15 A.M.	Key Note Address:-	-	**Dr.M.Y.Kawoosa** HOD, Chest Disease
	VOTE of Thanks		Representative of CIBA GEIGY. (Mr.Sajad Hussain)
Tea Break			
	Chairman	:	Dr.M.Y.Kawoosa.
10.30 A.M.	**Dr.G.M.Malik** Associate Prof.Head GIT Division Medicine Deptt.G.M.C.Sgr.	-	Unusual presentation of cases T.B.of Oesophagus, Liver, & Rectum
11.A.M.	**Dr.Gh.Rasool** [illegible]	-	Drug Resistance in T.B.
11.30 A.M.	**Dr.Quyoom** Residant	-	Epidominology of T.B. in Children
12 -	**Dr.Javid** Residant	-	AIDS & T.B.
1.P.M. -	**LUNCH**		

This symposium was sponsored by Medical Education Division of CIBA Geigy Ltd. Kashmir Branch.

Deptt. of Medicine, Government Medical College Srinagar.

Hospital in Sept.1994 A Case of Jaundice

Course & Curriculum

The conventional curriculum would need a review where emphasis be laid on problem oriented teaching and community aspect of medicine. In addition to:

1. Bed side teaching and out-patient teaching and emergencyserive in hospitals. Students be exposed to work in rural setting's in a primary health centre. Problem solving and patient care should be more practical and solutions demonstrated to students

In curriculum seminar symposium, groups discussions, tutorials, Clinico-pathological conferences, clinical meetings, inter departmental conferences, mortality conferences be encouraged.

The medical school library should be:

- Updated
- Issuing facilities improved
- Sorting system modernized or computerized with Medline system for references.
- Loose leaf text book system introduced
- *Newer teaching methods* like computer, T.V., telephone Dictaphone, projections, epidioscope be used.

Research programme : be made mandatory and compulsory

This would include incentive for research.

Promotion based on research ability and capability, special award and incentive to research scholars.

Thesis system be more research oriented than a mere formality.

Assessment

The assessment of medical students be: Constant

With an objective to create:

1. Awareness of ignorance in student by continuous assessment.
2. Suggesting means to compact inadequacies.
3. Assessment be more individualized.
4. The performance score should reflect in the quality of teacher and teaching in a medical school.

Panel Discussion on G.I.T. Motility Disorders at Sgr.
(R-L: Dr. Malik, Dr. Khuroo, Dr. Allaqaband, Dr. Durani)

"A Profile of GI Disease in the Valley"

Hamid Ali Durani ,M.D. Ex-Prof. & Head GIT.Division SMHS Hospital Sgr.

Gastrointestinal (GI) disease constitute an important cause of morbidity and mortality in Kashmir Valley. A resume of the more common and important ailments with reference to my experience of past thirty years is being presented in this manuscript.

Duodenal Ulcer Disease (DUD): one of the commonest ailment, is now know to the lay people as a chronic recurring disease with periods of exacerbation and also the important cause of melena and hometemesis. Such class of people as business executives, doctors, lawyers and academicians, 'earn the ulcer and nurse it', and know that 'the ulcer is the prize of success'. I have seen more dormant ulcers presenting with activation and becoming aggressive and refractory during the past five years of increased stress in life situations. Such observations will sure play down the place of the high profile H.Pylori as the sole causative agent and the infective theory of the genesis of duodenal ulcers, inspite of the plethora of the documentary evidence published in favour of H Pylori as the causative agent. It appears to over-simplify the genesis of DUD. There are other important factors, viz., the genetic factor the hypersecr extory status and the all important factor of chronic stress. On the treatment side, we can drive satisfaction from the fact that the present day therapy at our disposal heals almost 90% of DUs, only a small percentage of patients with non healing ulcers or those with complications like receurrent bleeds or stenosis come in for surgery.

Three ***GI Cancers*** are commonly diagnosed, Gastric Carcinoma, Esophageal Carcinoma and Rectal Carcinoma, in that order of frequency. The prevalence rate of Gastric and Esophageal Cancers in the valley is the highest in India.

Gastric Cancers : are seen more frequently in the males, located at i) cardia or pyloric antrum as localised tumor masses (the endemic type) and ii) as diffuse carcinoma in the body of stomach (the epidemic type), the later being galloping and rapidly fatal with blood borne metastasis. Carcinoma Cardia and Pyloric Carcinomas are more frequently diagnosed than the diffuse carcinoma[3]. Large majority of Gastric Cancers are Adenocarcinomas of different varieties, the tumor is not radiosensitive. Late reporting is the main reason for the infrequent endoscopic and histologic diagnosis of ***Early Gastric Cancers***[3], thus negating the chances of five year survival in majority of patients after gastric resection. It will be appropriate to remind that in Japanese hands, the 5-year and even 10 year survival of resected Early Gastric Cancer is in the range of 90-95%.

Esophageal Carcinomas are picked up with almost equal frequency, most often located in mid esophagus. The squamous cell carcinoma is radiosensitive, but surgical resection followed by radiotherapy is the accepted standard therapy, the overall outlook is gloomy, the average survival being in the range of 18-24 months.

The analysis of the etiological factors of gastric and esophageal cancers will require a lengthy write-up. One can sum up by stating that such carcinomas may result from the interplay of several factors, such as environmental factors and dietary habits and many more, targeting on a high risk population[2,3].

A host of syndromes: some recognized others ill defined, symptoms pertaining to GI tract or abdomen and physical examination drawing a blank, form a large chunk of patients, where diagnosis is generally forthcoming, but therapy challenging at times frustrating broadly this is the ***Functional GI Syndrome,*** related to long standing stress or as a part of depressive illness.

A comparative mention can be made of two extremes of functional GI disorders, vis., the ***Anoresis Nervosa*** patient who is grossly asthenic, undernourished female with depression and virtual refusal to eat, and the very ***Obese Compulsive Eater***, again a female who is otherwise cheerful and healthy but has a host of complaints entirely non-specific.

The best known of the functional GI disorders is the Irritable Bowel Syndrome, diagnosed precisely not by exclusion of organic disease but by its own presenting features, viz., the personality, the mannerism, the long story sometimes handwritten, which finally sums up to 3 or 4 bowel movements with a stereotyped pattern and emphasis on passing or loosing mucus in stools with added chronology of plenty of `gas' getting in the head, the story is supplemented by the statement of loss of `vigour'. Inspite of the exhaustive documentation of subjective symptoms, the patient is generally well fed and has not lost weight. The choice of therapy is plenty. One will have to use his skill and experience in devising a prescription for each patient taking into account the patient's stress on symptomatology and his likely acceptance of drug regimen.

The syndromes related to upper GI tract include ***incessant belching or vomiting***, the ***agenblase syndrome, globus hystericus*** and ***Esophageal chest discomfort(ECP)***. All require a careful evaluation and a lot of reassurance by way of therapy to allay the phobia of heart disease, organic esophageal disease or peptic ulcer disease.

An interesting syndrome seen in young girls with main presentation of repeated bouts of hemetemesis of long duration but with absence of melena. Symptoms like esophageal discomfort, regurgitation of food or unrelated vomiting of even a glass of water, are often described in addition to the main symptom of hemetemesis. Surprisingly there is no anemia inspite of the long history of hemetemesis, the appetite is good and patient looks healthy. Physical examination reveals no abnormal findings, except features of hysterical behaviour or anxiety in some. Upper GI Endoscopy, if performed immediately after the bout of hemetemesis, reveals longitudinal mucosal tears in the lower end of esophagus which heal up in a few days time. This group of patients, of which I have under my care about a dozen patients, should be labelled as cases of ***Primary Mallory Weiss Syndrome*** as originally described by Mallory and Weiss. It is the effort of compulsive vomiting which causes the esophageal mucosal tears. The syndrome is difficult to treat, prolonged psychotherapy and reassurance will be the mainstay of any therapeutic regimen for such patients.

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1 Clinical Courier August 1990 8(9). International Congress on Optimal Control of Gastric Acidity, Perth, Western Australia, 1990

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Knifeless Surgery

"Catheters Replace Scalpels in Many Operations"

Dr. F. A. Shawl M.D[*]

To reach the damaged heart valve, cardiologist threaded the tiny tube through a vein in the leg and into the right side of the heart. He then poked the tube through the muscular wall that divides the heart in half to reach the damaged valve on the left side.

Patient L.V. 38, was lying on the table, awake and watching an X-ray TV screen as the tube punched through his heart muscle. "I imagined I could feel the guide wire pass through the chambers of the heart," L.V said at a Washington Adventist Hospital press conference the day after the procedure. "It was uncomfortable, but not excruciating."

Once into the left side of the heart, doctor pushed a thin, deflated balloon into the opening of a mitral valve damaged by rheumatic fever presumably suffered as a child. The damage prevevted the valve from opening fully, choking L.V's blood flow, and threatening his life. With the balloon inplace, Shawl inflated it, breaking the narrowing obstructions and restoring the blood flow.

For L.V. the first procedure of its kind performed in the Washington area not only saved his life, but also allowed him to avoid the trauma and expense of openheart surgery.

For patients all over America, similar procedures are increasingly taking scalpels out of the hands of surgeons and replacing them with techniques that allow physicians to operate without incisions.

The trend is based on two technical advances: improved imaging systems and increasingly sophisticated catheter technology.

With new viewing systems, including enhanced X-ray equipment, CAT scanners, magnetic resonance imagers and ultrasound, physicians can peer into the body spot diseases they previously could not see.

Once the problem is found, a physician can reach it with a catheter-a slim flexible tube that can be steered through the labyrinth of the body's blood vessels. Catheters also can be passed over stiff but flexible wire guides (much like a guitar string) punched through the skin to reach the diseased area.

Catheters can take tissue samples, remove blockages, plug leaky vessels, infections, deliver drugs to a specific site and implant shunts to keep open passageways like the bile duct.

Several physicians said this is a rapidly advancing field that offers the promise of less quicker recovery, and less cost than the surgical techniques it replaces. "You substitute a needle stick for a skin incision," said Dr.Klemens H. Barth, director of vascular and interventional radiology at Georgetown University Medical Center. "There is no question that this is taking business away from surgeons."

"The field of general surgery is being invaded on a number of different sides by the less surgical techniques," agreed Dr. Haroid Coons, director of interventional radiology at sharp Memorial Hospital in San Diego. But "it is not a fight for turf.... There certainly are things but surgeons can do that we wili never able to do" by remote control.

That could change, however, if remote control procedures become available for such bread-and-butter operations as gall bladder removals, Coons said. General surgeons remove 200,000 to 600,000 gall bladders a year. Physicians are working on ways to destroy the gall bladder using catheters alone.

Surgical Substitutes

Doctors used one of the first forms of remote control surgery to eliminate the swelling of President Reagan's prostate. Instead of opening his abdomen to reach the walnut-sized organ at the neck of the urinary bladder, his physicians inserted a catheter through the urethra and into the swollen gland to remove the excessive material.

The president was back at the White House in three days, though the total recuperative period is excepted to be six weeks. If he had undergone abdominal surgery, recovery woulu have taken much longer.

Large, flexible tubes with fibre optic viewing systems also allowed physicians to detect the president's colon cancer a year and a half ago and perform routine follow-up exams every six months.

As experience with these technologies has grown, specialists have adapted them to their own needs. Sports doctors routinely perform arthroscopic surgerydone through viewing scopes poked through the skin-to repair damaged ligaments and smooth torn cartilage.

---

[*] Director Cardiology in Washington Adventist Hospital Deptt.Maryland USA.

Gynecologists routinely use a laproscop-a fiber optic scope stuckthrough the abdominal skin-to open blocked fallopian tubes, collect eggs from the ovary for in vitro fertilization or to destroy displaced endonetrial tissue from the ulterus.

Probably the best known example of the revolution is the switch from open-heart surgery to reroute blood around obstructed coronary arteries to the use of tiny balloon catheters. The catheters reach into narrowed coronary arteries and restore normal blood flow by crushing back the obstruction. As a result, bypass surgery is down 30 percent at Washington Adventists Hospital, Shawl said.

And that has a big impact on cost. Open heart surgery to fix a mutral valve costs $30,000. The balloon procedure cost only $5,000.

New Access to Organs

Minimally invasive surgery also has given physicians access to parts of the body that they previously could not reach.

The liver, for example, is a vast, spongy network of blood vessels and bile ducts that can become obstructed by a gall stones, atherosclerosis, cancer masses. Catheters can reach deep within the liver, a place surgeons cannot reach without removing huge chunks of the organ, to relieve the obstructions.

In the brain, arteries sometimes merge directly with veins, forming large, pulshing tangles that frequently cause intensive pain and may rupture, causing a stroke. Surgeons can remove many of these malformations, but some are beyond their reach, said Dr. David O.Davis a neuroradiologist at The George Washington University Medical Center. Sometimes, these tangles can be reached with catheters threaded through the arteries of the brain, and then shut down by injecting various materials into the defective connections.

Not all of the catheters are used through blood vessels. Abscesses, pockets of infection that may arise spontaneously anywhere in the body, or may be caused by an infection from previous surgery can be treated by pushing a catheter through the skin and into the abscess. Once pierced, the abscess can then be drained by a catheter, and the infection cleared up.

Sound and Light Surgery

The use of lasers is beginning to revolutionize even this revolutionary field of minimal surgeries. Once of the widest uses is in ophthalmology where lasers shot through the open pupil can weld shut bleeding arteries that threaten to peel the retina of the back of the eye, causing blindness.

Lasers have been used experimentally in arteries the blast through clots in the leg and the heart that cannot be fixed with balloons. The laser light is directed down a catheter filled with glass fibres that can both be used to look inside the vessel and aim the laser beam.

Since technology has evolved so far that no tool is put into the body at all. Kidney stones, for example, used to require extensive surgery to open the body, open the kidney and remove the stones. Urologists developed techniques for sticking a tube through the skin and into the kidney to remove the stones or

to break them up with ultrasound. Now, kidney stones are crushed by putting the patient in a bath tub and then pulsing focused shock waves at the body to break up the stones.

The revolution has not given flawlessly. There clearly are limits and side effects even from minimal surgeries. And those pushing the technology are strugging to set standards for when to use the techniques and to reach these standards to other physician. Several physicians said the general public also does not know that the alternatives exist.

'It is important for people to realize that these procedures are in competition with surgery," said Georgetown's Barth Sometimes, the alternative might be better.

[illegible] in India

Dr. Bashir Ahmad Gash. M.D

[illegible]

Bombay from Hong Kong introduced it for the frist time in India, killing more than 13 million people. There have been recent pandemics, the most serious of which raged intermittently for 30 years, costing millon lives in India.

Rodents are chronic carriers of plague and infection is spread from rodent to rodent by means of rat fleas (Xenopsylla). In India , wild rats constitute a perenial source of infection, since they form a permanent reservior for the preservation of the Yersina. Epizootics among rodents commonly precede the human epidemics, and this was amply proved on the recent outbreak. The national press has highlighted the fact that basic health workers in Beed district of Maharashtra duly reported an increase in rat fall as early as August, followed by a report on the unusual flea swarming. Seemingly the high officials ignored the warning. The results were 60 precious lives and an unseen panic all over the country. The lessons to be learnt were that in the science of epidemiology no base level information is too trivial to be overlooked, and that a strict blockade of the area is warranted in every suspected plague epidemic.

Intensive extermination of rats and fleas, better sanitation and intensive public cooperation are the mainstay of prevention of plague, sporadic as well as epidemic, and all the beyond the control of the health officials, who at the most could have a marginal role in health education.

Months have passed since the Indian epidemic of Gujarat rocked the world, and a new battle has now ensued. This time among the microbiologists at many leading institutes of the country. Recently, while the world was relaxing its embargo on the 'PLAGUE-INFESTED" India, the experts at the B.J.Medical College (Pune) claimed to have isolated Psuedomonas Pseudomallei from 10 samples received by it, leaving the National Institute of Communicable Diseases alone in backing the plague theory. Epidemiologically and clinically, no distinction is possible between plague and melioidosis, the s0-called 'WITHMORES DOSEASE'. Melioidosis, FIRST DESCRIBED in RANGOON, has been endemic in the sub-continent for centuries, and the confusion is probably forgivable except for the repercussions the 'BLACK DEATH' phobia and for the prestige and economy of India.

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Medical Treatment of Bleeding Peptic Ulcer

"Old Drugs, New Regimens"

Hemorrhage from peptic ulcer is due to the erosion of artery at the ulcer base by the combined digestive actions of acid and pepsin, platelet plug and clot formation seal the bleeding artery. Gastric acid has a detrimenta effect on the stability of clot as a $pH < 7$ results in inhibitionl of platelet aggregation and dissolution of clot[1] 'and intragastric acid prolongs the duration of bleeding[2]. Moreover the acidic gastric juice contains fibrinolytic substances[3]. Dissolution of the clot is the most important factor for peptic ulcer rebleeding[4] which require emergency surgery. Understandably then, attempts to counteract the acid (by H_2 receptor antagonists, antacids or both[5] or somatostatin[6]) or pepsin (by pepstatin[7]) or to inhibit fibrinolysis (by tranexamic acid[8]) should result in stabilization of th e clot and prevention of rebleeding. Yet till date the efficacy of none of the above mentioned drugs has been demonstrated unequivocally in the treatment of bleeding peptic ulcer[4], although meta-analysis of the available data suggests that H_2 receptor antagonists may improve morbidity and mortality from upper gastrointstinal hemorrhage.

Recent studies have suggested a close link between effective control of intragastric acidity and gastrointestinal bleeding. High dose ranitidine (600mg BD) has been shown to protect effectively the human gastric mucosa from the injurious effect of aspirin (as evidenced by a reduction in the intragastric hemorrhagic lesions), an effect which could not be achieved while using conventional doses of ranitidine[9]. Similarly omeprazole (most potent suppressant of gastric acid known) has been shown to decrease the incidence of active bleeding in patients with acute upper gastrointestinal hemorrhage[10]. One of the important reasons mentioned for lack of efficacy of conventional medical therapeutic regimens in control of bleeding from peptic ulcer has been their inability to adequately control intragastric acidity by failing to maintain sustained intragastric achlorhydria[11] experimentally shown to be essential for stabilization of clot[1]. Peterson et al[12] have shown that sustained achlorhydria can be achieved only with hourly intravenous bolus injections of cimetidine (100mg) alongwith a continous nasogastric infusion of an antacid at the rate of 0.5ml/min. and not with conventionally used doses of H_2 receptor antagonists with or without antacids.

In a preliminary prospective randomized study in patients with bleeding peptic ulcer using the above mentioned regimens we have recently shown that not only could suatained achlorhydria be maintained but also a higher rate of control of bleeding than that obtained with the standard regimes could be achieved[13]. Furthermore using such a regimen for all subsequent patients with bleeding peptic ulcer we achieved an overal control of bleeding in 75.4% of patients as compared with 56.7% in the historical controls[14].

To sum up we feel that there is increasing evidence that suggest a relationship between intragastric acidity, clot formation and peptic ulcer bleeding. Suprisingly however, there has been no large study of patients with bleeding peptic ulcer having being treated with a intensive medical regimen[13] aimed at complete neutralization of acis and pepsin with the intent of preventing clot dissolution and rebleeding. Since such a regimen is inexpensive, easily available and safe, we think there is an impending need for evoluating its efficacy in a large number of patients admitted with bleeding peptic ulcer.

ANIL A
(M.D.D.M.)

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(MD,Ph.D)

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Intra Coronary Stents

"State of Art in 1995"

Upendra Kaul*

During the last decade, percutaneous transluminal coronary angioplasty (PTCA) has become a very frequently used coronary revascularization procedure. Success rates generally exceed 90% despite the rapid expansion into new indications which inculude unstable angina, acute myocardial infamiction, more complex lesions and multivessel disease.

PTCA however is still limited by significant problems which have not significantly come down despite improved hardware and increasing operator experiences. These are abrubt vessel closure during or shortly after the procedure (2%- 9%), inability to cross or dilate some lesions esp chronic total occlusions (5%-10%) and a disturbingly high rate of restenoses (30%-45%). The procedure of intra coronary stenting has been developed to address some of these problems.

BAIL OUT STENTING

Intra coronary stenting to treat a major intimal dissection leading to accute threatened closure or acute occulusion is also termed as 'bail out stenting'. Although acute occlusion occuring during or within 24 hours of PTCA occurs only in a minority of procedures but is associated with a high rate of serious complitions with a reported mortality of 2-10%, emergency CABG rate of 20-70% and Q wave myocardial infraction rate of 20%-80%.

Since intimal tearing is the most frequent operative mechanism for this major complications the role of an intra coronary scaffold in the form of a mettallic stent has been used as an approach for sealing intimal flaps and enlarging the coronary lumen after balloon induced intimal disruption with a very high success rate of 90-96%. Focal dissections (10-15mm) leading to accute threatened closure appear to be ideally suited for placement of a single stent. Longer dissections if treated with stenting will require placement of more than 1 tandem stents depending upon the length of the disrupted segment.

Since certian morphologic lesions characterestics have been shown to be associated with a high rate of intimal disruption and subsequent vessel closure, the planned use of the stent for these type of lesions may lead to improved primary angioplasty outcomes compared with standard balloon dilation. In our centre we prepare these patients for stent implantation and proceed for angioplasty. If the result is satisfactory the post PTCA protocol is followed. However if the result is sub-optimal a stent is deployed.

Guidelines for Emergent Stenting after failed PTCA

1. Determine cause of failure if possible a. Avoid stent use if primary vessel thrombosis b. Consider stent for dissection or elastic recoil.
2. Maximize present anticoagulation a. Ensure that aspirin was given Pre-PTCA b. Administer heparin to ACT > 300s. c. Administer LMW dex tran (10%) as a continuous infusion (100 ml/h)
3. Anatomical considerations a. Loca tion, size, length, tortuousity, side branches of target site b. Ease of delivery, choice of guide cathe ter c. Number of stents to complete ly seal dissection d. Minimize residual stenosis.
4. Poststent considerations a. Pro longed angiographic observation (30-45 m). 1. Aggressive treatment of intrastent thrombus.

   2. Consider early surgery if subop timal stent result.

   b. Careful anticoagulation manage ment.

Intra-coronary Stents - Technical Features

	Wallstent (Medinvent)	Palmaz-Schatz (Johnson & Johnson)	Glanturco-Roubin (Cook)	Wiktor (Medtronic)
Design	Self expanding	Balloon expandable	Balloon expandable	Balloon expandable
Configuration	Woven wire	Slotted tube	Flexible	Flexible
Material	Stainless steel	Stainless steel	Stainless steel	Tantalum
Strut diameter (mm)	0.08	0.08	0.15	0.13
Delivery sheath	Yes	Yes	No	No
Length(mm)	15-30	15	15-25	15

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    1. Minimize heparin interruption for sheath removal
    2. Keep PTT high (> 100 s)
    3. Overlap heparin with therapeutic PT for 48 h
c. Prolonged in-hospital monitoring

De-nova Stenting

The deployment of an intra coronary stent electively in a focal lesion (de-nova stenting) in vessels more than 3 mms in size has been shown to produce very good acute results with almost negligible residual stenosis, very low rate dissections and acute closures. As per the "bigger is better" hypothesis proposed by Donald Baim, this should reduce restenosis as well. A number of non-randomised studies have shown a low rate of restenosis after de-nova stenting (10-15%).

Recently two large multicenteric studies. BENESTENT trial from Europe & STRESS trial from USA have confirmed that as compared to balloon angioplasty deployment of intra coronary stent in large vessels reduces the 6 months angiographic restenosis rate (22% vs 32% in BENESTENT trial & 32% vs 42% in STRESS trial). Both the studies have shown that the acute gain is significantly more in the stent group and despite a greater late loss the net gain is significantly more in this group as compared to the balloon angioplasty. This translates into a lower restenosis rate.

Technical Considerations:

Coronary stenting is a technically demanding procedure which requires as a pre-requisite, considerable knowledge, skill and experience in conventional coronary angioplasty. Although the technique is not very different from standard PTCA but the procedure & instruments are less forgiving and there are a number of unique considerations & difficulties.

Delivery Difficulties:

The delivery system's diameter is substantially larger than the conventional angioplasty balloons. Consequently it must be delivered through a guide catheter with a minimum diameter of 0.079 inches. Gianturco-Roubin stent 3.5 & 4.0 mm size needs a even larger guiding catheter with an inner diameter of more than 0.090 inches.

The system is substantially less trackable than a conventional angioplasty balloon catheter.

A perfect co-axial alignment of the guiding catheter tip & the target vessel is very important. If a balloon mounted stent (unless it is protected by a delivery sheath as in Palmaz-Schatz stent) after coming out of the guiding catheter tip cannot be negotiated across the target lesion it cannot be deployed. The whole system which includes guiding catheteri has to be pulled out. An appropriate selection of the guiding catheter is therefore very important.

Requirement for Accurate Deployment:

Once deployed a stent cannot be repositioned.

Positioning Considerations:

1. It is undesirable to place a stent across a major side branch. This is especially true for a Plalmaz-Schatz stent because of its design unlike the coil stents like Gianturco Roubin stent.
2. If multiple stents are to be placed the degree of overlap should be minimized.
3. The delivery in multiple stents should be always proceeding from distal portion to proximal portion.

Anti-Thrombotic Therapy for Stent Implantation

Preprocedure

Aspirin 325 mg per day including the day of the procedure.

Ticlopidine 250 mg BD being at least the evening before the procedure.

Warfarin 10mg the evening before the procedure.

10% Low-molecular weight dextran I.V. at 100 ml/h. beginning at least 4 h before the procedure.

Intraprocedure

Continue 10% Low-molecular weight dextran I.V. at 50ml/h.

Heparin 10,000U I.V. with supplementation as needed to maintain activated clotting time greater than 300 s through the procedure.

Early Postprocedure

Initially do not continue heparin unless plans are to delay catheter removal. Restart heparin 2 hours following catheter removal wihtout a bolus. Adjust infusion rate to achieve a PTT between 60 s and 90 s or ACT 140-150 sec.

Continue 10% Low-molcular weight dextran at 50 ml/h until a total dose of 1000 ml has been administered.

Continue aspirin and ticlopindine.

Warfarin 10mg the evening following the procedure.

Last Postprocedure

Continue warfarin to achieve PT 16.0-18.0 (INR 2.5-3.0)

Maintain PT in that range for 30 days postimplantation.

Continue I.V. heparin until PT is greater than 20.0 for 24 h (INR 2.5 to 3).

Continue aspirin indefinitely.

Continue ticlopidine for 3 months.

Coronary Stent Thrombosis

One of the serious limitation of stent deployment has been the occurence of acute & subacute stent thrombosis. The frequency of this complication has been as high as 25% in earlier series with a majority of them occuring wihtin 2 weeks of the atent deployment. The mortality of stent thrombosis is significant and can be as high as 7-10%. If the stents are to achieve their promise of optimizing the primary angioplasty result & reducing the restenosis risk the issue of thrombogenicity must be adressed.

Our understanding of pre & post stent antithrombotic protocols & better methods of delivery of stents with post stent balloon expansion at high pressure have significantly reduced these problems in the last few years. The current acute & subacute thrombosis rates after stent deployment in high volume centres is < 5%.

Predictors of Subacute Thrombosis Following Intraccronary Stenting

Inadequate anticoaqualation
Intraprocedural thrombus before or after stent
Residual stenosis higher

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Dissection present poststent
Residual intimal dissection
Eccentricity
Residual distal vessel irreqularity poststent
Bailout indication
Unstable angina
Type-C lesion
Lesion length > 1.5 cm
Plague area > 3.5 cm
Symptomatic post-PTCA dissection
Incomplete wrapping of dissection

Peripheral Vascular Complications:

The intensive antithrombotic treatment along with use of relatively large introducing sheaths etc increase the groin complications & bleeding complications following stent deployment.

Local Entry Site Complications:

The groin complications include haematoma, pseudoaneurysm & arteriovenous fistula formation. The incidence varies between 2%-15%. However most of these complications can be minimised by adhering to rigid post stent for 48 hours & protocols use of c-clamp compressions. The advent of radial artery stenting which utilises radial artery puncture & use of crimped stents on standard balloon catheters deployed through 6 french guiding catheters has minimised these problems & allowed rapid ambulation.

Bleeding Complication:

These include G.I. Bleed retroperitoneal haematoma and cerebrovascular accidents etc. These are the result of intensive antiplatelet & anticoagulant useage.

A meticulous control of anticoagulation with avoiding excessive doses of heparin or oral anticoagulants has helped in reducing these complications to less than 5%. > The recent data from Europe suggest that after proper deployment of a stent using post deployment high pressure balloon inflation, the requirement of anticoagulation does not exceed 7-8 days. This is especially true in elective stenting with vessels more than 3.25 mms in diameter.

Future of Stents

With recognition that thrombogenecity by regrowth of endothelium preliminary studies evaluating the seeding of intravascular stents with genetically engineered endothelial cells have been performed. In addition an improvement in stent funcition will be realised with the locally delivered anticoagulants, thrombolytic or antiproliferative molecules.

EXPERIENCE OF PALMAZ-SCHATZ STENTING AT AIIMS, NEW DELHI

Between September 1993 and December 1994 we have mplanted Palmaz-Schatz corronary stents in 85 patients. Fifty patients presented with stable angina and 29 with unstable ngina. Stenting was carried out for denovo, focal lesion in arge coronary arteries (N=53), significant dissection during TCA with acute threatend closure (N=16) and saphenous vein raft stenosis (N =2). Succesful delivery of the stent was chieved in 84 cases. Percent diameter stenosis was reduced rom 77±11% to ±8% amd minital liminal diameter increased from 1.08±0.28 mm to 2.92±0.39 mms. There was 1 death due to left main dissection secondary to guiding catheter trauma in a patients taken up for a bail out stenting. One patient of oral anticoagulants succumbed to a head injury 2 weeks after discharge from the hospital after successful bail out stenting. Local vascular/bleeding .pl66

complications occured in 43 patients (4.6%). There was no instance of acute or subacute stent thrombosis. Sixty patients are symptom free. Sixty months angiography study is planed in all. Twenty five patients who have already undergone it, have demonstrated restenosis (>50% diameter reduction) in 2 and a new lesion distal to stent in 1. All have been successfully redilated.

A high rate of successful deliver of the Palmaz-Schatz coronary stent can be achieved in wide spectrum of patients with very few complications. Long term results are very gratifying & encouring.

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Quinolones and Macrolides:

''Rolesin RespiratoryInfections''

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Newer agents in these classes can be useful when standardantibiotics may be contraindicated or ineffective. They are well tolerated gastriontestinally and have excellent tissue pentration when give orally, allowing shoter courses at lower dosages. Quinolones are especially effective against nosocomial infections, macrolids against community-acquired infections.

The new fluorquinolones and macrolids are potent antimicrobials with considerable potential to solve some difficult problems and case management of respiratory tract infections. Offering a broad range of activity against most of the common repiratory tract pathogens, the drug have their major impact on infections of the lower respiratiry tract where drug coverage had been unavailable. The new quinolones are particularly effective against most of the pathogens typically involved in nosocomial respiratory infections. The macrolides, which also are effective against upper respiratory infections, are especially appropriate for community-acquired infections(Table I).

Two new macrolides have been approved recently and others are currently under development. Various quinolones have been making their appearance since the 1960s. Because clinical experience is greater with them than with the macrolides, this discussion will emphasize recent findings with quinolonès.

Nalidixic acid was the frist of the quinolones to be developed, but in its oral from it lacked sufficient tissue penetration to perfrom well in urinary tract infections, for which it was designed. Never quinolones-norfloxacin, pefloxacin, enoxacin, ofloxacin, lomefloxacin, and ciprofloxacin- have excellent tissue penetration when given orally. Ciprofloxacin was approved for respiratory tract infections in the mid-1980s, and ofloxacin and lomefloxacin were approved in the past year.

The new macrolides arose from numerous development efforts to improve erythromycin, which has been used since 1950 to manage uncomplicated community-acquired infctions. Altering the chemical configuration of the macrolide ring has resulted in agents superior to erythromycin in many respects, including bioavailablity, gastric acid stability, gastrointestinal toleranece antibacterial activity, peak levels in serum after oral administration, tissue concentration, and serum half-life.

Clarithromycin and azithromycin are the two new macrolides available for treatment of respiratory infections. Clinical trials are in progress to determine the efficacy and safty of various other recentlydeveloped macrolides, including roxithromycin, midecamycin, josamycin, and spiramycin.

Quinolones and macrolides have different pharmacokinetics and modes og action. Quinolones act at the DNA level, interfering with replication and thereby killing the bacteria. The macrolides interfere with bacterial protein synthesis. Both classes of agents are effective against a wide range of bacteria. Both are also distinguished by excellent tissue penetration, achieving very high levels in the lung parenchyma. Drug concentrations in the bronchial tree, the interstitium of the lung, and the alveolar spaces are much higher than serum concentrations. As a result, therapy can be shorter and dosages less frequent than is possible with other antimicrobial agents. One of the quinolones, lomefloxacin has been approved for once-a-day dosing.

Table 1. Quinolones and Macrolides Approved for respiratory tract infections

	Primary Use
Quinolones Ciprofloxacin (IV and oral)	Against gram-negative, aerobic organisms Lower RTI in elderly patie Nosocomail RTI Acute exacerbations of COPD RTI associated with alcohois diabetes, cystic IV and oral therapy
Ofloxacin (IV) and Oral)	Against gram-negative, aerobic organismsNosocomial RTI Acute exacerbatio of COPD RTI associated with alcohoism, diabetes, cystic fibrosis Sequential and oral therapy
Lomefloxacin (oral)	Acute exncerbations of COPD caused by Hemophlius influenzae and Mora catarrhalis

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Macrolides Clarithromycin (oral)	Upper RTI, including acute maxillary sinusitis Pneumonias caused by streptococcus pneumoniae and MYcoplasma pneumoniae Acute bacterial exacerbations of COPD caused by S. Pneumomiae, H.influenzae, or M.catarrhalis
Azithromyacin (oral)	Acute bacterial exacerbations of COPD caused by S.pneumomiae,H.influenzae, or M.catarrhalis (oral) Community-acquired, outpatient pneumonias caused by S.Pneumoniae or H.influenzae

COPD=chronic obstructive pulmonary disease

Bioavailability, which ranges from 70% to 95% ciprofloxacin and ofloxacin, which are available in both oral and intravenous froms, also offer the possibility of sequentail therapy. The hospitalized patient can be started in intravenous therapy. The hospitalized patient can be started in intravenous therapy and then switched in two or three days to oral therapy. This can sorten hospitalized, hold down the cost of antibotic therapy and promote better patient compliance. The never macrolides currently are available only or in oral from.

Clinical efficacy of Quinolones

Numerous pathogens may be resposible for respiratory infection and these may vary from time to time and place to place generally, however, a handful of pathogens are responsible for most such infection. In patients with chronic bronchitis or asthma, acute bronchial infection ussually result from Hemophilus influenzae, H.parainfluenzae, Streptococcus pneumoniae, Moraxella (Branhamella) catarrhalis, and Neisseria sp. Less often involved are Klebsiella sp. Pseudomonas sp. Staphylococcus aureus, Serratia marcescens. and various streptococci. A 1987 study by Sanford Chodosh at Boston University showed that four pathogens accounted for almost three fourths of the acute bacterial infections in patient from whom 171 bacterial species were isolated. H.influenzae accounted for 24% of case, H. parainfleunzae for 17%, S.pneumoniae for 19.9%, and M.catarrhalis for 11.1%.

In treating serious respiratory infections, physicians often are faced with the problem of begining therapy before laboratory findings are available. Foe such empiric therapy, the choice of a macrolide, quinolone, or other antimicrobial agent is best guided by the setting in which the infection is encountered. In nursing homes, for instance, the incidence of gram-negative pneumonias among elderly patients is high, and quinolones are attractive because they cover most, if not all, of the common gram-negative pathogens.

Of the three quinolones available for respiratory tract infection, ciprofloxacin has been clinically tested most thoroughly and has proved to be effective against a broad range of respiratory infections (Table I). In a double-blind clinical trial of 87 patients will bacterial bronchitis, we compared oral ciprofloxacin with ampicillin for safety and efficacy. Both drugs achieved excellent results, with ciprofloxacine achieving 98% and ampicillin achieving 89% disappearance of signs and symptoms. Ciprofloxacine was well tolerated, with few adverse effects and significantly lower incidence of diarrhea than with ampicillin.

We also assessed the safety and efficacy of ciprofloxacine in 40 patients with community-or hospital-acquired pneumonia. All but one of them had chronic under lying lung disease. Pneumococci were the most common pathogens identified, accounting for 29% of isolates. Hemophilus sp accounted for another 24%. The results were similar to those of other clinical trials:A clinical cure was established-disappearance of symptoms, clearing the chest film, and reduction of high leukocyte counts- in 12 of the 14 patients Treatment side effects were in significant.

Serveral studies have reported on the effectivencess of oral ciprofloxacin in treating bacterial infection in cystic fibrosis patients. Whose respratiry problems are most commonly exacerbated, S.aureus, and H. influenzae.

There has been some question whether quinolones are effective against S.pneumoniae. Laboratory studies indicate that the minimum inhibitory concentration at which ciprofloxacin is effective against S.pneumomiae is quite high, perhaps as high as 4 ug/mg. Because of this investigators logically concluded that S. pneumionae infection will not respond to quinolones in a clinical setting; one very early clinical study (which,however, appears to have been flawed) seemed to bear this out. Studies scince then have produced different results; for example, a group of several studies , including two we did at Nassau County Medical Center, showed a cumalative cure rate of 92% (59 of 64 patients). In these studies, the minimum inhibitory concentrations for S.pneumoniae in some cases were quite high, as in those earlier in vitro studies, but the levels were unrelated to clinical effectiveness. The explanation probably lies in the drug's pharmacokinetics: Ciprofloxacin achieves very high lung parenchymal and bronchial tissue penetration. Which could explain the apparent contradiction between in vitroresistance and clinical effectiveness.

We have also found that ofloxacin can be effective against S.pneumomiae infection in a joint study. We found that ofloxacin therapy led to cures in eight of 10 patients who had bacteremic S.pneumoniae infections.

Even when both laboratory and clinical studies are taken into account, it is still appropriate to use penicillin for uncomplicated, community-acquired S.pneumoniae in an otherwise healthy persons. However, when the patient has S.pneumoniae and underlying chronic pulmonary disease, when the infection is complicated by alcoholism or diabetes, or when it occurs in a nursing home setting, there is a high probability that other pathogens, such as H. influenzae, will be present. In such cases, the quinolones

Table 2, Sensitivity of Common Respiratory Tract Pathogens to some Quinolones and Macrolides

	Sensitive	Intermediate	Resistant	Insufficient Data
Ciprofloxacin	Moraxella Catarrhalis Hemophilus influenzae Klebsiella sp Neisseria sp Pseudomonas aeruginosa Serratia marcescens Staphylococcus aureus	Legionella sp Mycobacterium tuberculosis Streptococcus Pneumoniae	Anaerobic Cocci Bacteroides sp Pseudomanas cepacia P.maltophilia	Chamydia sp Mycoplasma sp
Ofloxacin	Klebsiella sp H.influenzae Bordetella pertussis Legionella pneumophila M.catarrhalis S.marcescens S.aureus (methicillin-resistant S.pneumomiae M.tuberculosis	P.aeruginosa Pseudomonas maltophilia S.pyoenes Listeria monocytogenes Mycobacterium aviumintracellulare Bacteroides fragils Peptostreptococcus sp	Nocardia asteroides Fusubacterium	
Lomefloxacin	M.catarrhalis E.coli S.aureus H.influenzae Klebsiella Pneunoniae Legionella sp L.pneumophila P.aeruginosa S.marcescens	Mycoplasma hominus M.pneumoniae	P.maltophilia P.cepacia B.fragilis S.pneumoniae	
Clarithromycin	Streptococci,including S.pneumoniae Staphylococci M.catarrhalis L.pneumophila Mycoplasma pneumoniae Chlamydia pneumoniae Borrelia burgdorferi	H.influenzae	Streptococci and Staphylococci resistant to erythromycin	
Azithromycin	H.influenzae M.ctarrhalis L.pneumophila C.pneumoniae M.pneumoniae B.burgdorferi	Stapylococci Streptococci Aerobic Facultative gram-negative bacilli	Streptococci and Staphlococci resistant to erythromycin	

are a good choice-to eradicate those other pathogens are very likely to be effective against S.pneumoniae as well.

When to Consider Quinolones

Given their well-documented clinical efficacy against specific pathogens, quinolones can be considered for a well-defined population (Table 2). Ciprofloxacin is a good choice for bacterial exacerbations of COPD. It is useful in managing lower respiratory tract infections in elderly patients, setting in which gram-negative organisms are Staphylococcus species are frequently patgogenic. The pharmacokinetics of ciprofloxacin also make it an attractive option in treating elderly patients. Although age may create an unfavorable terrain for absorption of the drug in the gastrointestinal tract, studies have demonstrated that serum concentrations in the elderly are similar to those in younger patients is useful in cystic fibrosis patients with respiratory infections. Not only is it effective against the three most common pathogens in these cases, it has additional advantages in its safety, oral bioavailability, and appropriateness for outpatient use. To lower the risk of emerging resistance, it is recommended that cipro-floxacin be alternated with other agents from infection to infection in cystic fibrosis patients.

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Because they are available in both intravenous and oral from, quinolones can be used in sequential monotherapy, with the patient receiving intravenous therapy for the frist few days and then oral therapy as the clinical profile improves. This is a monotherapy from of antibiotic streamlining, the process of transferring patients from complicated, equally efficacious, and less expensive regimen.

A cost-analysis of the anribiotic streamlining program at Hartford (Conn.) Hospital showsthat the cost of antibiotic therapy per patient is significantly lowered through the use of sequential intravenous and oral therapy, primarily because patients can be discharged earlier. Early discharge also minimizes the risk of nosocomial infections and may reduce the nosocomial pressure on microbial flora within the hospital. At hartford hospital, it has been found that difficult-to-treat oeganisms such as Pseudomonas emerge as resistant pathogens more frequently during prolonged in patient antibiotic therapy.

Tabel 3. Antimicrobial Agents and Treatment of Respiratory Tract Infection

Antimicrobial	Consider for	DO NOT
Quinolones	Bacterial exaceberations of COPD Elderly patients likely to have gram-negative infection Lower RTI in diabetics and alcohol abusers Lower RTI in cystic fibrosis patients	Consider for Children and Pregnant Woman' Anaerobic bacterial respiratiry infections
Macrolides	Upper RTI, including sinusitis and laryngeal and oral cavity infections Acute bacterial exaceberation of COPD RTI in otherwise healthy patients, including community-acquired pneumonia	Gram-negative infections other than Hempohhilus
Penicillins	Lower RTI known to be caused by S.pneumoniae	Moraxella catarrhalis Hemophilus influuenzae Most gram-negative bacilli
B-Lactamase inhibitors	Streptococci Staphylococci (except methicillin-resistant)	Methicillin-resistant S.aureus Gram-negative bacteria

'Except in life-threatening situations where other alternatives are not effective

Quinolones are expensive, as are the new macrolides. If the patient has an infection that can be treated effectively in the home with penicillin, then the cost of quinolones or macrolides is not justified. Butin properly selected cases, both are cost-effective. For penicillins-resistant or comlicated infections, and for very sick patients who must begin their therapy in the hospital, the cost of quinolones or macrolides is justified. Our analysis at nassau Country Medical Center showed that we saved thousands od dollars by using sequential ciprofloxacin therapy with hospitalized patients who could be discharged realtively quickly to continue oral therapy at home.

In some well-defined populations quinolones are not the drug of choice. they should not be consider when the pathogens are likely to be anaerobes, thich are only moderately sensitive to quinolones -for example, in aspiration pneumonias. Nor, as previously indicated are quinolones the drug of choice for otherwise healthy patients known to have an uncomplicated S.pneumoniae infection.

Quinolones should be used sparingly in children and are contraindicated in pregnat woman. because animal studies have shown that the drugs can interfere with cartilage growth. However,quinolones have been used in life-threatening situations in which nothing else was available for-example, for Pseudomonas pmeumonia in young cystic fibrosis patients. In this instance, the use of quinolones appears to be as a safe as it is in adults.

Several other precautions should be kept in mind. Gastrointestinal absorption of the fluorquinolones can be reduced by the concurrent administration of aluminum- or magnesium-containing antacids, zinc in multivitamin preparations, oral preparations of iron, and high-dose calcium supplements. Sucralfate also interferes with absorption of quinolones.

It has been shown that quinolones slow theophylline metabolism and can rise the patient's theopylline level signifcantly. This interaction varies with the different quinolones. Enoxacin increases theophylline levels the most, ciprofloxacin has less effect, and ofloxacin in the least. We have not onbserved theophylline increses when the dose of ciprofloxacin is less than 1 gm per day. However, if the patient is elderly and has long been on the ophylline, it is prudent to monitor theophylline levels and, if needed adjust the dosage, to minimize the risk of toxicity.

The safty, board spectrum of activity, clinical efficacy, and convenience of an antibiotic clearly do not obviate the need to use cliniacl jedgement. Approximately 24% to 66% of antibiotics prescribed in the united States are thought to be unnecessary or inappropriate. In 1989, almost half of the $2.4 billon spent on orally administered antibiotics in the United States was for five new, expensive agents: ciprofloxacin, norfloxacin, amoxicillin-calvulanate potassium, cefaclor. In most instances a breif clinical assessment may reveal a less expensive or more appropriate drug for the infection under consideration.

When to consider Macrolides

We have had less clinical experience with the newer macrolides, which have been aproved for use in respiratory infections just in the past year. They have been shown to be effective against upper respiratory tract infection, which are often caused by gram-postive bacteria . The macrolides can be used to treat sinusitis, oral cavity infection, and laryngeal infections, although older, lessexpensive agents such as ß-lactams or sulfonamides are also effective against these infections.

For lower respiratory tract infections, macrolides join the quiolones as one of the two most attractive classes of agents, with the macrolides having the edge in treating community-acquired respitatory infections in young, otherwise healthy patients . They are recommended for outpatient treatment od community-acquired pneumonias, which are commonly caused by S. pneumoniae or H. influenzae. The new macrolides are also active against other community-acquired pathogens such as Chlamydia. Legionella, and Mycoplasama. Clarithromycin and azithromycin are both effective against acute bacterial exacerbation of chronic bronchitis caused by S.pneumoniae, H.influenzae, or M.catarrhalis.

In the past, ß-lactams or penicillin could be considered for treating patients with community-acquired lower respiratory tract infections, but they may be ineffective when the infection involes Hemophilus sp, which are often resistant to ampicillin. Most of the previously available drugs other than erythromycin are ineffective against Leggioella. With the new macrolides, all the common community -acquired respiratory pathogens are covered. And with their high levels of tissue penetration, the new macrolides , like the quinolones, are effective when given just once or twice a day.

Aside from Hemophilus, coverage of gram-negative bacteria by macrolides is not very effective. The agents, therfore, are not indicated when gram-negative infection is suspected,as, for instance, in elderly patients.

Unlike the quinolones, the macrolides have not been found to interfere with cartilage growth in animal studies. However, their safty and efectiveness for pediatric use have not been established. There is no evidence that microlides interact with antiacids . The major

concern with erythromycin has been patient tolerance. Intrvenously administered erythromycin can cause phlebitis; the oral from may produce serious GI upset. The new macrolides have fewer side efects, probbly because they have increased gastric acid stability and thus do not generate toxic matabolites. Some investigators have observed interaction between clarithromycin and theophylline, but this is infrequent and does not often appear to be clinically significant.

Duration and resistance

The duration of treatment with both equinolones and macrolides is an open question. conventional therapy for pneumonia was established over three or four decades with penicillin. sulfonamides, and ß-lactams. Traditional, patients are treated for one to two weeks . Macrolides and quienolones are significantly different classes of medication, achieving higher levels in the lung than do other agents, so it may not be neccesary to treat respiratory infections for as long. Urinary tract infections can be resolved with these drugs in two or three days, and respiratory infection may need to be treated for only three to five days. Clinical studies are needed to established the appropriate dueration of treatment, an increasingly important consideration in the current economic climate.

Resistance is a major concern with all new classes of antimicrobials. The mechanism by which quinolones work gives us reason to be optimistic about their continued effectiveness. Because they interfere with bacterial DNA replication, there is a little concern that resistance will be transferred from one bacterial to another by plasmids. Nonetheless, several centers have found that some organsims, particularly methicillin-resistance S.aureus, have developed resistance to equinolones. The new quienolones and macrolides, like all new antimacrobials, should be used judiciously to minimize the likelihood that resistance will develop.

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Social Profiles of Child Health in 3rd World Countries''

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SOCIOMEDICAL PROFILE:

With the establishment of a primary health care infrastructure, emphasizing on health promotion and health protection, using inexpensive and appropriate health technologies, relying on health auxiliaries and community volunteers, and deriving support from intersectoral co-ordination India opted for a need-based, cost-effective system conforming to her socio-cultural milieu. Thus was laid the foundation for primary child care at the grass roots level. Subsequently, with the change over from 'family planning' to 'family welfare', the emphasis shifted towards maternal and child health; family planning services started being offered as a component of a health package including maternal care, child care, nutrition and health education. These changes were necessitated to eliminate coercion, on the one hand, and to ensure child survival, on the other, so that contraceptive practices are adopted by the people voluntarily. With these policy changes, a favourable administrative and operational frame work was established which is conductive to the health and welfare of children. The movement of 'Health for All' gave a further boost to child health. Universal immunization and oral rehydration became the main stay for child survival throughout the developing world. ORT, which is currently available to one in three children suffering from diarrhoea, saves about 3000 lives per day, and more than 1 million young lives a year. The decade intervening between 1980s and 1990s witnessed a rise in the coverage of children under immunization from a humble 20% to almost 80%. It indicates the existence of a health infrastructure that is capable of reaching out to 100 million infants, several times a year, and distributing the benefits of scientific progress to population at their door steps. Universal immunization succeeded in creating a massive dent in the vaccine preventable childhood diseases in the developing world to the extent that mortality reduced from 2.5 million to just over 1 million in measles; from 700,000 to approximately 400,000 in pertusis; from 1.1 million to less than 600,000 in neonatal tetanus; from 19000 to 4000 in diphtheria; and in polio myelitis the annual incidence of residual paralysis decreased from 400,000 to almost 140,000. The under-five child mortality from vaccine-preventable diseases in the developing countries, declined from an overall toll of 11.6 million in 1983 to 8 million in 1992. The mortality is expected to reduce further during the succeeding years. An equally significant contribution of immunization is a substantial reduction in the threat to nutritional health and long-term growth of children posed by vaccine-preventable diseases. Pneumonia, another major killer of under-five children, registered a marginal mortality decline from 3.3 to 3.1 million deaths in the same period; the proposed acute respiratory infection control programme has not yet been implemented by the developing world countries. Child survival rate (the percentage of children who survive to the age of 5) has been growing in the developing countries. While India has a child survival rate of 84.8% it is 95.5% in China and Sri Lanka, 83.1% in Pakistan and 80.9% in Bangladesh. Singapore and Cuba, having child survival rates of 98.8% and 98.1% respectively, are leading the developing countries in this respect.

SOCIOCULTURAL PROFILE

A child is a centre of attraction in the Indian social life receiving all the love and affection, care and attention, not only, from parents and grandparents, but also, from kiths and kins of a traditional Indian family. Strong sociocultural ties exist between children and their parents; more specifically between mothers and their children as a consequence of strong bonding attachment reinforced by breast-feeding and intimate physical and emotional contact.

Breast-feeding is a universal practice in Indian families, especially in rural areas, and is continued day and night, right from the birth of a child. Some mothers, having misconceptions about colostrum, discard colostral feeding which deprives the infant from all its nutritional and non-nutritional benefits. This practice has been particularly studied in some African tribes where, under the belief that colostrum is poisonous, newborns are wet-nursed or fed on animal milk, local drinks or even herbal preparations, at least, till the mother is able to bring forth the matured milk. pregnancy, delivery and puerperium may be as natural to rural women in developing countries as breast feeding carrying no anxiety or tension beyond an inconvenience in the normal routine of life. Resting and feeding up of mothers for a period after delivery is a healthy Indian practice favourable to successful breast-feeding. However, dietary restrictions in pregnancy and puerperium, which may be observed in certain traditions, are detrimentel to maternal and child health; so is the delayed and inappropriate weaning and restricted feeding of children when suffering from fever or diarrhoea.

Harmful traditional practices, adversely child health, are common in third world countries. Such practices, which are usually based on superstitions or misconceptions, may be seen among ignorant, tradition-bound rural masses. Branding of children is but one example of a traditional practice occasionally observed in Indian villages. Hot iron rods are applied to skin to produce a burn-mark just to 'word off' evil spirits and 'bring in' health and welfare to children.

Under the pressure of changing circumstances the traditional joint family system has started breaking in cities and towns of this country yielding place to nuclear families which, despite their sosioeconomic advantages, are by and large, ill-equipped to stand the stress and strain of modern life. Self-centered and materialistic living, associated with the nuclear family system, not infrequently results in marital discord and parental conflicts with devastanding consequences on children. The nuclear family system is associated with an increase in lactation failure and a corresponding decrease in the bonding effect. With the collapse of joint family system instances of spouse abuse. Child abuse and substance abuse have started multiplying, threatening the life and survival of children. Smoking, drinking and drug addiction are social evils that bear an inverse relationship with child health, especially if indulged in by expectant and nursing women. Various unhealthy cultural practices like smoke-emiting open cooking processes, defaecation in open fields, bare foot walking on soiled grounds, water collection from step-wells etc. are casually associated with acute respiratory infection, hook-worm and guinea-worm infestations, and a variety of water-borne and filth-borne diseases afflicting children more than the adult population.

Discrimination against female children in Indian families has deep-rooted cultural background whereby, while a son is considered as an asset, a girl is an all time liability. A son receives preferential treatment on the grounds that he carries forward the family lineage; posesses economic potential; can support the family and attend to parents in their old age; and can perform religious rites and ceremonies as and when necessary. On the contrary, a girl is treated as an economic burden, a symbol of dowry system and a property of some other family of no use to her parents in their old age. She gets a raw deal within her family in all respects including food, clothing and education. Discrimination of the girl child may start even before birth; antenatal sex confirmation may prompt the parents to ask for female foeticide. Unwanted girl children, in such families, may be candidates for neglect. abuse, infanticide or even suicide. sex bias may be, in some measure, responsible for the declining male to female sex-ratio's in this country. At the begining of this century, the female ratio per 1000 males stood at 972; it declined progressively to 930 in 1971. The census of 1981 showed a marginal improvement when the ratio was estimated at 935; subsequently, according to 1991 census, it further declined to 929. The decline in female population ratio is a sad reflection on Indian ethos and a subject of serious concern demanding speculation an introspection. Another kind of discrimination, having sociocultural roots in Indian society is the inbuilt caste system.

SOCIOECONOMIC PROFILE

The categorization of the countries of the world into industrialized, developing and least developed groups is essential based on socio-economic norms as reflected in their widely varying per capita G.N.P values estimated at 18884, 843 and 240 US$ for the industrialized, the developing and the least developed countries, respectively. The socioeconomic level of a country broadly determines the health status and the life-style of its population. The allocations made on health and education vis-a-vis defence reflects the health consciousness of governments; their awareness of the interdependence of health and development; and their concern for the health and well being of their people.

A review of the three catagories (TableII) brings out that industrialized countries spend 13% of government expenditure on health, 4% on education and 13% on defence, equating health with defence. The developing countries spend 4% on health, 11% on education and 11% on defence. The least developed countries spend 5% on health, 13% on education and 13% on defence. Thus on the basis of outlays, with education and defence are held at par, health is neglected both by the developing and the least developed countries. Choosing life expectancy at birth and the low birth-weight percentage as the two important indicators of community health, the impact of socioeconomic development of countries could be clearly demonstrated. We find that the life expectancy at birth is 76, 61, 50 years and the low birth-weight frequency is 6%, 9% and 24% in the industrialized, developing and the least developed countries respectively. The health scene of the industrialized countries is a pointer to the fact that socioeconomic progress promotes health which, in turn, promotes further socioeconomic development. But that should not imply that health cannot be achieved without adequate socioeconomic progress. Developing countries like China. Philippines, Egypt etc., having a per capita income below $1000 a year, have achieved the target of reducing the under-five mortality rate to 70 per 1000 births. Some of the sub-Saharan African countries have reduced malnutrition rates below 15%, which is less than half of the average rate for the developing world, as a whole.

Nearer home, a comparative study of the socioeconomic profile of India, Pakistan, China and Sri Lanka (TableI) and its impact on the health and health-related areas, on the one hand, and the outlays on health and education sectors vis-a-vis the defence sector, on the other, may be quite revealing. In terms of per capita GNP, India occupies the lowest place and Sri Lanka the highest. The socioeconomic gradient of these 4 countries is reflected in their life expentancy at birth which is 60 years in India, 59 in Pakistan and 71 years in China and Sri Lanka. A comparative analysis of other parameters reveal that Inadia is leading the other countries only in respect of safe water supply but lags behind the rest in sanition and the percentage of low birth-weight deliveries. China leads in sanitation and Sri Lanka in adult female literacy. On the whole, the populations in China and Sri Lanka enjoy better facilities in terms of health care, education and environmental sanitation.

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Within India itself, the state of Kerala has succeeded in achieving the highest standards of health in this country, despite socioeconomic constaints that it shares with other states. A comparison between the statistical indices of Punjab (the richest state in the country), Kerala (having average per capita income below the national average) .col

Table I: Socioeconomic Progress in India, Pakistan. China and Sri Lanka with Special Reference to Community Health

Indicators	India	Pakistan	China	Sri Lanka
GNP per capita (US $).	330	400	370	500
Population %age below absolute poverty level (1980-89).	Urban 29 rural 33	Urban 32 rural 29	Urban - rural 13	Urban - rural -
%age of Govt. expenditure on health (1986-92).	2	1	-	5
On education (1986-92).	2	2	-	8
On defence (1986-92).	17	28	8	9
Life expectancy at birth (1992)	60	59	71	71
Low birth wt.%age (1990)	33	25	9	25
Population %age with access to safe water(1988-91)	85	56	72	60
Population %age adequate sanitation (1988-91)	16	24	79	50
Population %age with acess to health services (1985-92)	-	55	90	93

Source: 'The State of the World's Children', 1994. (UNICEF).

Table 2: Socioeconomic Progress in Industrialized, Developing and the Least Developedcountries of the World

Indicators	Industrialized countries	Developing countries	Least developed countries
G.P.N. per capita (US$).	18884	843	240
Population percentage below absolute poverty level:			
Urban	-	27	55
Rural	-	31	70
Percentage of government exoenditure			
On health	13	04	05
On education	04	11	13
On defence	13	11	13

The State of the World's Children', 1994. (UNICEF).

TABLE 3: Important Health Indices of the States of Punjab and Kerala in Comparison of the All India Values

Indices	Punjab (inter-state rank)	Kerala (inter-state rank)	All India
Infant mortality rate (1989).	64 (3)	21 (1)	91
Crude birth rate (1989).	28.3 (6)	20.3 (1)	30.6
Couple protection rate (1991).	75.8 (1)	55.6 (6)	44.1
Mean female marital age (1981).	21.1 (2)	21.8 (1)	18.3
female literacy (1991).	49.7 (6)	86.9 (1)	39.4

Source: 'Action Plan for Revamping the Family Welfare Programme in India.'
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and India, as a whole, may be quite revealing in this respect (Table I). It may be observed that the Kerala leads all other states in the country in having the lowest infant mortality and crude birth rates, as also, in possessing the highest female literacy and female marital age. The achievements of Punjab, though much better than the national average, exceed those of Kerala only in respect of couple protection rate. Kerala has earned the distinction of being the healthiest state in the country, despite its low per capita income. The healthiness of Kerala and its healthfull behaviour has been attributed to its high female literacy. The other factors of significance are that Kerela has succeeded, to a large extent, in striking a balance between the preventive and curative care, on the one hand, and the urban and rural services, on the other, Kerala has paid concentrated attention on MCH care, Punjab is not lagging far behind.

SOCIOPOLITICAL PROFILE

The awareness that children are an important national asset and the fulfilment of their health and nutritional needs is fundamental to national progress, is an essential pre-condition for lying the foundations for health care of children in any country. This realization, which is shaped by the sociopolitical climate of a country, is steadily growing in the developing countries and has been mainly instrumental for their signing of the Alma Ata declaration and accepting the challenge for providing 'Health for All' and formulating national programmes of action for meeting that challenge. Since 'Health for All' goals can be achieved independent of expensive professional services imposed on developing countries that may be handicapped by the financial implications of primary health care if 20% of their governmental spending and 20% of their overseas aid budget in basic education, safe water supply, basic health care and family welfare. These fundamental services which are highly costeffective shall, for obvious reasons, promote health and well-being of children, more than any other segment of population. A universal achievement of these goals is likely to prevent 2 million child deaths annually besides of protecting millions of other children from disabling states like blindness and deformities.

Political will for child health is not lacking in this country. Provisions for promotion and protection of child health and well-being are enshrined in the constitution of India. Promotion of child health is contained in the article 47 providing for 'raising the level of nutrition and the standard of living of its people and the improvement of public health'. Protection of child health is contained in the articles 39, 41 and 42 envisaging 'protection of childhood and youth against exploitation and moral and material abandonment; and a suitable climate for 'humane conditions for work'; as also a social welfare system capable of offering 'public assistance in cases of unemployment, sickness and disablement' as well as 'for maternity relief.

Political commitment of the government of India was reflected in it's sighning of the Alma Ata declaration on primary health care; laying adequate stress on maternal and child health services; and accepting the challenge of providing 'Health for All' by the year 2000.

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Ramadan Fasting

"Effects on Health and Disease"

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Ramadan is the ninth lunar month of the muslim Year during which it isobligatory for all healthy adult muslims to observefast from dawn to sun-set[1].Fasting in Islam means total abstination from eating, drinking and having sexual intercourse from dawn to dusk for the whole month Of Ramadan[2]. People may eat or drink at night, but not during the day. After sun-set they take a meal called "Iftar" and the last meal they consume is "suhur" just before "Fajar" (dawn) prayers. The food during Ramadan is basically similar to the ordinary food consumed during rest of the eleven months except that a large quantity is taken at "Iftar" and at "suhur",instead of several meals during the day. There is a tendency of consuming more of drinks and sweat foods at or after Iftar. The sick, those travelling, pregnant, lactating and menstruating women are permitted to pospone their observation of Ramadan fast till some other suitable time[3]. The doctors have been concerned about the effects of Ramadan fasting on different physiological parameters as also on various disease states. Many studies have been conducted so far in this regard, the results of which are briefed in a following few lines.

Body compartment and Heart rate

There is no significant change in the mean body weight, body mass index , skin fold thickness, mid-arm circumference, total body water and fat body mass during Ramadan fasting[2]. However, some have observed a decrease in body weight,

bodymass and subcutaneous fat among both sexes, especially females, if the energy intake is reduced during fasting[4]. Further, some have noticed that there is a significant increase in caloric fat, carbohydrate and protein intake during fasting, thereby, causing a sigificant increase in weight during Ramadan[5]. Also, it is suggested that isotonic saline ingestion, perhaps by careful ditetary manupulation, may reduce fluid losses and be of value for those, who observe fast in humid hot weather and that too for many hours.[2] No change has been detected in the urine output and fluid intake during fasting. A greater reduction of heart rate has been noticed in males as compared to females.[4]

Biochemical parameters

It has been observed that serum cholesterol, triglycerides, total proteins, albumin, alkaline phosphatase, chloride, phosphorus, glucose, Blood urea, creatinine, uric acid, glutamate oxaloacetate transaminase (SGOT) and glutamate pyruvate transaninase (SGPT) show a significant increase during the fasting period. These parameters come to base-line as the fasting month is over.[1,6,7] However, some have noticed that if the caloric intake is reduced during fasting, there occurs a significant decrease of Blood triglycerides and high density lipoprotein-cholesterol (HDL-chol.) but low density Lipoprotein-be increased in this study[8]. Further, some have found no change in serum cholesterol, triglycerides and HDL-chol. during the fasting month.[2,6,9] A highly significant decrease of serum magnesium, sodium and corbon dioxide and a highly significant increase of potassium, glycose and calcium has also been reported in fasting individuals. However, no change has been noticed in the various kidney parameters (after taking non-steroidal anti-inflammatory drugs) in the fastinf individuals when compared with controls[2]. It has been recommended that patients of hypertension, hypercholesterolaemia, hyperglycemia, hyperiricemia and heart, liver and kidney diseases require both careful medical consultation before continuing to fast and supervision during the fasting period[1].

HAEMATOLOGICAL PARAMETERS

Haemoglobin level, red blood cell count and serum iron show a slight decrease, while as, an insignificant fluctuation has been reported in case of packed cell volume (PVC), mean corpuscular volume (MVC), mean corpuscular haemoglobin (MCH) anf mean copuscular haemoglobin concentration (MCHC)[1]. However, a significant change has been detected in case of mean platelet volume (Which decreases) and platelet distribution (which increase) in fasting individuals. There occurs no harmful effect on red cell survival during the fasting month. Futher no change has benn observed in the coagulation profile (i.e prothrombin time, partial thromboplatin time, thrombin time, reptilase time, plasma fibrinogen, antithrombin III, platelet count, factor VIII, clotting activity, vonwillibrand's factor, ristoctin co-factor, factors-V,VII, and X, plasminogen and _$_2$ antiplasmin) during the fasting month, when compared to the study on a non-fasting day. However, platelet aggregation respose to adenine disphosphat (ADP), adrenaline, collagen and arachidonic acid is diminished in fasting individuals. Futher, it has been suggested that patients on oral anticoagulation therapy should observe the fasting without feeling any danger of incerease in thromboembolic and haemorragic complications[2].

Immunity

A significant improvement in cell mediated immune responses (as indicated by an increase of helper-inducer "T" cell count, helper cell/suppressor cell ratio, cell functional index, total system functional index and pokeweed MIT index) has been onservedduring Ramadan fasting of the immunoglobulins, Ige is moderetely increased. These preliminary results indicate a favourable effect of islamic fasting on immunity[2].

Risk of obesity and Islamic Fasting

Obesity is a known risk factor for coronary heart disease, diabetes, Hypertension, gall bladder disease, gout, respiratory disease etc. futher, obesity increase the risk during the surgical intervension and the post-operative period, as also there is increased incidence of certain cancers. Islamic teaching participate in solving these problems by prohibiting drinking alcohol, eating pork and its products (rich in fat) and cell for slopping smoking. Islamic fasting, in particular, decreases the risks of obesity by shaping the body's demands as per its needs and keeps a control on frequent and excessive eating[2].

Endocrine System

No significant change has been detected in the fasting levels or half an hour after food in case of serum gastrin and insulin, when compared to the levels on a non-fasting day[6]. However, variation in the timing of peak levelsin case of prolactin and insulin has been observed, occuring at 9PM during Ramadan fasting as compared to 4PM and 4PM for the respective hormones on a non-fasting day, while as, no such variation has been detected in the timing of lowest levels of these hormones. Also, no significant fluctuations have been noticed in case of cortisol during Ramadan fasting, when compared to the levels on a non-fasting day. On the basis of above observations it has been concluded that Ramadan fast, on the whole, is not a stressful procedure, besides, the peak levels of insulin at night in Ramadan, as has been suggested, may be of the potential benefit for a better control of a maturity on set diabetes. Ramadan fasting, as onserved, does not alter the standard indices of throid function (i.e levels of thvroxine-F . triiodothyroine, thyroid stimulating hormone and Free thyroxin index) in healthy adult males[2]. However, some have noticed a sginificant increase of T_4 during fasting[6].

Male Fertility

Ramadan fast does not have any adverse effect on the fertility of healthy men,. In fact it has been suggested that fasting improves the total sperm count, the gonadotrophic hormone levels and the testosterone level in fertile males. Besides, fasting does not either improve or decrease the seminal quality of azoospermics, but may rather have some benificial effect on oligozoospermics by improving spermatogenesis either via the hypothalamo-pituitary-testicular axis or by a direct effect on the testis[2].

Diabetes mellitus

Diabetic patients, who are on a diet control only should find the Ramadan fast highly benificial, in case weight reduction is achieved by proper adjustment of calorick in take[10,11]. Several recommendations have been made for the patients of insulin dependent diabetes mellitus (IDDM). Some suggest only one suitable evening dose and ommission of the morning dose[12]. Some recommend twice daily insulin doses (as short acting insulin) comprising one-half or one third of their usual morning dose to be taken before "Suhur" and the usual evening dose before breaking their fast. the above recommendations, as advised, should be under proper medical suppervision [10,12].However, Ketosis prone or brittle diabetics have been particularly advised to aviod fasting during Ramadan[12]. Patients of non-insulin dependent diabetes mellitus (NIDDM) have been advised to reduce the usual daily dose of oral hypoglycemics to one third or one fourth, to be taken "Suhur". Among the hypoglycemic durgs, glibenclamide has found to be an effective and safe durg for the fasting NIDDM patients and the recommended regimen is to take usual morning dose toegther with any mid-day dose at sun set and any evening dose before dawn[13]. However, some have observed that fasting in NIDDM patients reduces the indegenous insulin relase and worstens hyperglycemia, thus requiring proper supervision to avoid any crisis[11].

Peptic Ulcer Disease

A preliminary prospective endoscopic evaluation of peptic ulcer disease during Ramadan fasting (1414 Hijri/Feb.-March 1994) was conducted on 23 volunteered fasting patients and 15 non-fasting controls at gastroenterology division of SMHS hospital, Srinagar a first study of this kind reported sofar. The patients (both fasting and non fasting) were endoscoped on the 7th day prior to and after Ramadan and were prescribed H_2-blockers (Tablet Ranitidne 150 mg twice daily) to be taken at "Suhur" and "Iftar", five fasting patients (22%) were drug defaulters. It was observed that 8 (89%) of the fasting chronic peptic ulcer patients (i, e chronic duodenal and chronic gastriculcers) bled during Ramadan and, unfortunately, all the five drug defaulters belonged to this catagory. From this study it was inferred that the chronic ulcers are difficult to hael during fasting. It was, thus, concluded that chronic peptic ulcer patients should not observe fast, though there was a limition only 23 patients volunteered for study and 5 (63%) of the bleeders were drug defaulters[14].

Urological diseases

There is a scaricity of data avaiable at persent in this regard. The effects of fasting on the patients of chronic uninary tract infection and stone formers have revealed a significant reducetion in urine volume, urinary calcium and increase in its specific gravity, when compared to the non-fasting group. Futher, a bighly significant increase in urinary sodium and potassium has been reported in these patients when compared to normal controls. However, non-significant changes have been noticed in acse of serum calcium, sodium, potassium,

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uric acid, creatinine, blood urea and in urinary creatinine, sodium and potasium in these patients. It has been suggested that some changes, in the form of decrease in urinary calcium and incerease in sodium and potassium in the above patients groupduring fasting, may actually be inhibitory to the stone formation[2].

Rhumatological Diseases

Fasting, by reducting overweighting, helps patients having degenerative diseases. Futher, fasting is considered to be a very helpful in the inflammatory diseasecs of autoimmune origin such as rheumatiod arthritis. Also, most of objective parameters of the varius rheumatological diseases (as observed by swedish researchers) are improved by fasting[2].

Fasting-its effects on Eyes

Fasting may lead to a state of relative dehydration leading to decrease in tearing and dry eye symptoms. In patients with border-line tears, fasting may lead to keratoconjuctivitis sicca. Adequate intake of fluids at "Suhur" and topical application of artificial tears may help reduce the signs and symptons. Patients are advised to occlude their puncta by pressure, which would prevent drainage of the drops to the throat, thus prevening breaking of their fast. Futher-more, fasting leads to decrease in glucose content of agueous, leading to change in the osmotic pressure of the enterior chamber. This may be followed by changes in the lens leading transient hyperopia. Understanding the basis mechanisims involved may help in the reassurance of such patients[2].

Besides, the studies on drug compliancef by patients during Ramadan fasting have revealed that majority of these do not follow the prescriber's instructions.[15,16] hus soma ill effects of Ramadan fasting on certain disease states can be attributed to this non-compliance.

The above mentioned observations may however, need to be capitalized on futher search and research, regarding the effects of Ramadan fasing on various physiological parameters and the disease states, to draw some conclusion in this regard.

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"Depressive disorders in Kashmir"-

A changing Socio-demographic and Clinical Profile of Patients over the past two Decades

Mushtaq A. Margoob*, A.A.Beg and K.S.Dutta*****,

ABSTRACT:

Demographic and clinical variables of 3468 new depressive patients who attended the Psychiatric Diseases Hospital, Srinagar during the three years 1971, 1980 and 1989 were compared. The profile of a typical patients seems to have undergone a marked change over the two decades. The depressive disorders have increased in general, with a marked increase in neurotic type of depression with preponderance of female patients from urban domicile. In addition to an increasing acceptance of this facility by the community and a better psychiatric awareness, the possibility of an actual increase in the incidence of depressive disorders has been discussed in this study.

Introduction:

Depression is one of the commonest psychiatric illness all over the world. It has been recognized, discussed, classified and treated since antiquity. The concept of depression, as viewed by layman and even most physicians depends upon the presence of a mood designated as sadness, despair, dejection, despondency, weeping and often associated with disturbed psychomotor activity in the shape of retardation or agitation; disturbances of biological functions like disturbances of sleep, appetite, body weight and sexual desire. If this pattern is not prominently seen, then for all intends and purposes the patients is not considered as being depressed. A number of reports have however, highlighted the fact that depressions may be disguised as various types of somatic symptoms, chron*ic pain, hypochondria and psychosomatic disorders[1,2] . The term masked depression

or depressive eqviualent imply that if one probes behind the psychosomatic symptons, one will find a depressive core from which an overt depressive affect or mood may eventually evolve. In many cases the depressive affect (sadness), weeping spells and dysphoria may not evolve at all. It is such cases which are likely to be misdiagnosed and treated for their somatic symptoms rather than treatment for the underlying emotional disorder. Hypochondriac symptoms, psychosomatic disorders and pain are the most common masks of depression among adults in the United States and in 1980 more than 10 billion dollars were spent on disability payment to patients with chronic pain problems alone[3]. The situation in the developing countries seems to be equally disappointing. An incidence of of depression among 41.9 per cent of medical wards of Rajindra Hospital of Govt. Medical College, Patiala[4]. In another study[5] of 80 patients of depression in Madras reported that somatic symptoms were the predominant presenting complaints in 61.3% of their patients.

Psychiatric diseases hospital, Sinagar, Kashmir in the sole psychiatric institute catering to the needs of emotionally disturbed patients of whole Kashmir Valley, adjoining district of Jammu Privince and a good number of patients from Ladakh as well. AS mentioned in an earlier study an analysis of data collected from such an institute can not only have important theoretical implications of an aetiological and cross cultural nature, but can be used to construct a client profile and to tailor and develop the services in an optional way so that the facilities available remain strictly relevant to the needs of the population. With this background in mind, the present study was undertaken to analyze some of the demographic and clinical aspects of our sample, especially in view of the tremendous increase in mental disorders (depressive disorders in particular) in Kashmir[7] over the past few years.

Material and Methods:

The sample for the study comprised of the case records of the following three series of the patients properly registered and treated at the Hospital for psychiatric Disease, Srinagar, Kashmir during the years 1971; 1980 and 1989 respectively. The reason for particularly selecting these three years for study during past almost two decades is that, the year 1971 is the first year when the process of proper maintenance of patients clinical profile and case records was started at the hospital and has been meticulously continued since then. The year 1989 was selected because due to prevailing disturbed conditions and migration of almost one third of the trained staff during the year 1990, had adversely affected (at least initially) the proper registration of each new patient treated at the hospital OPD. The 1980 is the mid year of the total time period under study.

Since classification of mood disorders is an area which is fraught with multiple controversies, an attempt has been made to present the results in a simple and straight forward manner as per WHO's classification, which has broadly speaking divided such disorders into two main groups of

(1) Endogenous depression : A more severe depressive disorder usually episodic and includes all entities like

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depressive psychosis, involutional depression or manic depressive psychosis depressive type and another grouping.

(2) **Neurotic depression :** a relatively less intense mood disorder and may run a continuous chronic course and includes term like reactive depression, depressive neurosis etc. All the patient included in the sample had been diagnosed by a consultant psychiatrist according to the international classification of diseases 8th and 9th revisions⁽⁸⁾, respectively.

Results and comments:

In the series of 3486 patients, our observations (Table I) show that the annual incidence of depressive disorders in the psychiatric population constituted approximately 13 to 31 per cent of all the psychiatric cases registered and the treated during the study period. Over the decades the percentage of depressive disorders has considerably increased in general but the neurotic depression or depressive neurosis has tremendously increased from 2.68 per cent during 1971 to 11.48 per cent during 1989. These figures are in complete agreement with the earlier studies of the authors[6, 9] and the study at PGI-Chandigarh[10]. An increase in the percentage of depressive patients could be due to increased awareness among the patients about the nature of such disorders and an imcreased acceptance of psychiatric intervention and treatment in such cases. However the increasing stress of the patient day life may be playing an important role in the increase of such disorders especially in case of neurotic or reactive depressive disorders as the stress is supposed to play by an important role in the etiology and psychopathology of these disorders. More and more people are adopting single family pattern, as in joint families both men and women could share their stresses and strain with the other members of the same sex, which is rather difficult in the single families. This argument finds further support when we compare our results with a European study conducted at Maudsley Hospital, London[11] who reported 24.6 per cent depressive neurosis in the series of 6433 psychiatric patients treated at the hospital during 1967-69. However an actual difference in the incidence of such disorders between east and west could not at the same time be ruled out[12]. A greatly increased percentage of female patients especially housewives, with almost an equally increased percentage from urban domocile could possibly be explained on the speculation that these urbal housewives go through an intense degrees of stress while performing the multiple duties of a

Table 1: Diagnostic profile of the sample.

Year of reg.	psychiatric patients registered reg. Total No (%)	End. dep. patients registered Total No (%)	Neu. dep . patients registered Total No (%)	depression patients registered Total No (%)
1971	1303 (100)	168 (12.89)	35 (2.68)	203 (15.57)
1980	867 (100)	83 (9.45)	37 (4.27)	119 (13.72)
1989	1298 (100)	260 (20.03)	149 (11.48)	409 (31.51)

Table 2: Socio-demographic variables of the sample.

			Sex				Age in Years		
Type of Dep.	Year of Reg.	No. of Dep.Pat. %(No)	Male %(No)	Female %(No)	Rural %(No)	Urban %(No)	upto 20 %(No)	21-35 %(No)	36- %(No)
ENDOGENOUS	1971	100 (168)	38.69 (65)	61.31 (103)	57.14 (96)	42.86 (72)	1.19 (2)	16.67 (27)	81.54 (137)
	1980	100 (82)	52.44 (43)	47.56 (39)	54.88 (45)	45.12 (37)	4.8 (4)	39.02 (32)	56.10 (46)
	1989	100 (260)	50.77 (132)	49.23 (128)	55 (143)	45 (117)	8.85 (23)	37.30 (97)	53.85 (1'140)

NEUROTIC	1971	100 (35)	22.86 (8)	77.14 (27)	28.57 (10)	71.43 (25)	8.57 (3)	62.86 (22)	28.57 (10)
	1980	100 (37)	29.73 (11)	70.27 (26)	32.43 (12)	67.57 (25)	8.10 (3)	54.05 (20)	37.83 (14)
	1989	100 (149)	40.27 (60)	59.73 (89)	30.20 (45)	69.80 (104)	14.09 (21)	51.68 (77)	34.23 (51)

wife, a mother and also manager of the house and hence fewer opportunities of giving vent to their emotional by restricting themselves with the four walls of the house. the other aspects of the patients profile seems to have remained almost constant over the years.

Keeping in view the present prevailing conditions, the study of the impact of the present stress on the nature of the emotional problems in general and stress related disorders in particular would be of great interest. The authors have started some basic work on the subject and the results are expected to be informative and useful one.

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A study on Combination of Praziquantel and albendazole in the treatment of hydatid disease

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Introduction

Albendazole is effective in repeated cources for the treatment of hydatid disease due to Echinococcus granulosus (1-4) but the response may take several months. Praziquantel has been shown to be effective against intestinal parasites, schistosomiasis, cysticercosis and other helminths (5,6,7). Praziquantel, when used as scolicidal agent in vitro and in vivo in animal models is found to be effective against E. granulosus (8,9,10,11). It has been suggested that praziquantel may be of value in the treatment of human E. granulosus infection (12). In human hydatid disease, treatment using combination of albendazole and praziquantel has not been used before. In this communication we report four cases, which were treated with a combination of albendazole and praziquantel with good response to therapy.

Material and methods

Treatment with combination of albendazole and praziquantel was tried on four patients with hydatid disease, who were investigated before treatment with complete haematological and biochemical blood tests, hydatid serology by indirect haemogglutination, chest x-ray, abdominal ultrasound and computed tomography scan. Monthly courses of albendazole were given and each course consists of albendazole 400 mg. B.D. for four weeks followed by two weeks off treatment. Praziquantel in a dose of 50 mg/kg was given in different regimens or the four patients either once daily, once weekly or once fortnightly or once a month. This regimen was given regularly till significant reduction in cyst size or disappearance of the cyst was observed.

Results and discussion

Table 1 describes the findings and outcome in the 4 patients. There was significant reduction in the cyst numbers and size or disappearance of the cyst following treatment.

Surgical or medical treatment of hydatid disease has never been satisfactory especially in disseminated form (14). Although albendazole has been found to be effective in some cases, the long duration of therapy (13) was necessitated the search for more effective therapy. In laboratory studies praziquantel even with low concentration of 50 mg/kg has shown to above scolocidal effect. All protoscoleces treated with praziquantel concentration of 100-500 mg/kg were found necrotic and dead in 12-18 days (Richards et al., 1988). In another experiment, proctos-colicidal effect of praziquantel was observed after 6 days, a much faster rate when compared to the albendazole (10). Also praziquantel efficacy in animal models in vivo an din vitro has been shown (8). Praziquantel is capable of penetrating the human hydatid cyst at a serum concentration of 1.3-1.8 m/l at a drug dose of 50 mg/kg (Patschke et al., 1979). The only human study on praziquantel in hydatid disease was presented from China.

We have studied the effect of albendazole on 22 patients with hydatid disease and during the mean follow up period of just over one year, in 4 patients (18.2%) the cyst completely disappeared and in another 5 patients (22.7%) there was a 30-60% reduction in cyst size (4). The present study was conducted as the response to albendazole was found to be slow and a combination of albendazole and praziquantel had not been used before. We used praziquantel with different regimens in combination of standard courses of albendazole. When compared with albendazole therapy alone (4) the response to the combination therapy was quick and effective in all four patients. Only one patient who was given praziquantel daily developed drowziness on the fifth day and disappeared when the dose was changed to once weekly. The hydatid cyst completely disappeared in three patients and most of the cysts disappeared in the fourth patient. The duration of therapy ranges between 2 - 3 months. In this preliminary human study with small number of patients and short follow up, the combination therapy was quick and effective, but further large studies are needed. The combination therapy might reduce the need for surgery in the future. Our aim is to increase the number of patients on this regimene and in parallel to this study with combination therapy, we are planning to study the effect of praziquantel alone for the treatment of Echinococcus. This will require a few months or may be years because of the relatively low number of patients with hydatid disease seen in Saudi Arabia.

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Table 1 **Case description**

Case No. age, sex and weih	History and findings	No. of cyst location & largest size	Albendazole	Praziquantel	Response to therapy hydatid titre before and after treatment
1 33/M 73 kg	Shortness of breath of two months O/E-NAD	12 Both lungs 1.5 x 3 cm 1 right lobe of liver 6 x 3.8 cm	a) 2 courses in itially b) 3 courses with praziquantel	a) Non initally b) Once monthly for two months then once weekly for on month	1. Disappearance of 1. ung cysts 2. Complete disappearaof liver cyst 2024 128
2 30/M 65 kg	Right hypochondrial pain for 2 years O/E-NAD	1. one Rt. lobe of liver 9 cm x 7 cm	2 courses	Once Weekly for 3 weeks for 2 months	Disappeared after treatment 512 64
3. 38/F 68 kg	Abdominal heaviness 3 operations for hydatid custs left lobe liver-15 y paraaortic-12 yr. left lung-10 yr. O/E - huge abdom. mass	1. Pelvis (one) 25 x 15 cm 2. Liver (one) 8 x 5 cm 3. Mediast enhum (three 15 x 6 cm	Albendazole 3 courses	Daily for 5 days the once weekly for 2 months	1. Size reduced to 5 x 3 cm after treatment, loss of daughter cysts 2. Disappeared size reduced to 3.3 x 2 cm 1024 64
4 50/M 76 kg	Right hypochondrial pain for six months O/E heapt omegaly	1. Rt. lobe Liver (one) 17 x 10 cm	Albendazole 3 courses	Once every two weeks for 2 months weekly for 1 month	Complete disappearance of daughter cysts and cystic nature 4096 256

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"Incidience and Management of Bell's Palsy."

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ABSTRACT:

Facial nerve paralysis is a source of considerable mental anguish not only due to the cosmetic deformity it induces but also due to difficulty encountered while eating and drinking. it deprives the patient of facial movements, causing the loss of facial expression which is enconscious reflection of emtions and moods.

One hundred and five patients of facial nerve paralysis of various causes were taken for this study. Out of these 80 patients wre diagnosed as cases of Bell's Palsy. These patients were divided into incomplete paralysis group and complete paralysis group. Because the treatment of Bell's Palsy remains a mystery as yet. In this study steroids, vasodilators and a placebo were compared with one another for treatment purposes and no difference was obtained with any form regarding the outcome.

MATERIAL AND METHODS:

The present study was conducted on 105 patients of facial nerve paralysis. Schirmer's test was done with porous paper strips 0.5 cm x 5 cm (blotting paper) placed over lower lid into conjunctival sac. The degree of wetting was recorded after 5 mitutes and 7 sides were compared. Reduction to 25 percent of normal side was taken as a significant degree of impairment and indicated lesions proximal to geniculate ganglion. Electrogustometry was performed by applying a galvanic current to tongue which evoked a metallic taste sensation. Lesion of facial nerve porximal to chorda tympani branch were associated with absent or decreased taste sensation.

Nerve excitability test was done by stimulating the nerve in the region of stylomastoid foremen. Threshold intensity was recorded and the difference in 2 sides recorded. No response to maximum intensity tolerated by the patient (20-30 ma for 0.3 msec) was indicative of degenerative lesion of nerve. Needle electromyography was performed bu inserting needle electrical activit at rest and at voluntary contraction. Recording of fibrilitations indicated possibility of incomplete cure as these potentials mean nerve fibre degeneration.

Recording of volunatary motor unit potentials and a polyphasic reinnervation potential indicated good prognosis.

TREATMENT:

Case of Bell's palsy were divided into 3 groups. One group received corticosteroids (prednisolone) a days according to following:-

80 mg/day for 5 days

60 mg/day for 1 day

40 mg/day for 1 day

20 mg/day for 1 day

10 mg/day for 1 day

Second group received vasodilators (Cyclandelate) in the dose of 800 mg/day x 9 days. Third group received a placebo (calcium tablets) for 9 days. Cases of facial palsy because of some other cases received the normal standard treatment according to disease.

OBSERVATIONS:

During a period of one year, 202450 patients attended S.M.H.S. Hospital, Srinagar for various aliments. Among them 105 patients had facial nerve paralysis giving an incidence of 51.86 per 100,000 patients.

Out of 105 patients, 80 patients (76.19%) were diagnosed as having Bell's Palsy, and so the incidence of Bell's Palsy is equal to 39.51 per 100,000 patients. Out of remaining 25 cases 10

(9.52%) had post-traumatic paralysis due to acute otitis media, 2 (1.90%) had Ramsay Hunt Syndrome and 2 (1,90%) infective polyneuritis. Leporsy, parotid malignancy and congenital facial palsy accounted for one case each (0.9%).

Bell's Palsy group:

This group consited of 80 patients. The age of patients ranged from 3 to 70 years. Howevre maximum number of patients were below age of 40 years. 52.5% patients were females and 47.7% were mailes. 51.25% patients had left sided paralysis and 48.75% had right sided paralysis.

45% patients had clinically complete paralysis and 55% had incmplete paralysis. level of lesion could be detected only in 75 patients as 5 patienta did not co-operate for tests. 7 patients had suprageniculate lesion. 55 patients had infrageniculate lesion and 13 patients had infrachordal lesion.

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Nerve excitability test was done in 75 patients. No difference in excitability threshold was found in 39 patients who had clinically incomplete paralysis. In complete paralysis group consisting of 36 patients 20 patients (55.55%) had no nerve excitability threshold difference. 5 patients (13.88%) had nerve exicitability threshold difference of more than 3.5 miliampers and 11 cases (30.55%) had no respose on maximal nerve stimulation.

Table No. 1
(Showing the aetiology for facial paralysis)

Cause:	No. of Cases	Percentage
1. Bell's Palasy	80	76.19
2. Post-Traumatic	10	9.52
3. Acute otitis media	8	7.61
4. Ramsay Hunt Syndrome	2	1.90
5. Infective Polyneuritis	2	1.90
6. Congenital	1	0.95
7. Leprosy	1	0.95
8. Parotid malignancy	1	0.95
Total	***105***	***100.00***

Needle EMG was done in same 75 patients. Patients have clinically incomplete (39) EMG revealed voluntary motor unit potentials in all cases while as patients having complete paralysis clinically. EMG revealed fibrillations in all patients. (30.55%). Motor unit potentials mixed with polyphasic potentails, and fasiculations were recorded in 25 patients (69.45%).

In complete parlysis group. 15 patients (34.40%) received steroids and had ful recovery. 9 patients (20.45%) received vasodilators, all recovered fully. 20 patients (45.45%) received placebo, all of then recovered fully.

In complete paralysis group, 17 patients (47.22%) received steroids and only 40 patients 82.35%) recovered. 3 patients had no improvement. 9 patients received vasodilators and only 8 recovered (88.88%). One patients (11%) had no improvemennt. 10 patients (27.77%) received a placebo and only 9 (90%) recovered. One patient had no inprovement.

The time taken to recover fully ranged 7 days to 12 weeks, though less in incomplete group (1 week to 6 weeks) than in complete paralysis group (3 weeks to 12 weeks), 5 patients who showed no signs of recovery till 3 months were in age group of 40-70 years. Four were hypertensive and one was diabetic. All the 5 had suprageniculate lesion. Nerve excitability tests showed no response on maximal stimulation and EMG showed fibrilation activity.

DISCUSSIONS

Taverner (1954) treated 26 patients of Bell's palsy with 1 gm of cortisone acetate given over a period of 8 days. He concluded that this treatment neither lessens the incidence of dennervation nor hastens the recovery[1]. In 1959 after studying 341 patients he concluded that there is no treatment of proved value[2]. Kumar et al, (1984) studied 34 patients of Bell's palsy and reached to conclusion that 73.5 percent patients recovered spontaconclusion[3]. May et al (1985) stated that bell's palsy is self limiting and spontaneously remitting disorder. 84 .pl66

percent patients recover satisfactorily without any treatment[4]. Prescott (1988) stuided 879 patients of Bell's Palsy and concluded that steriods did not influence the outcome[5].

CONCLUSIONS:

1. Incindence of facial nerve paralysis in this hospital is 51.86 per 100,000 patients.

2. Commonest cause of paralysis was found to be Bell's palsy. Incidence being 39.51 per 100,000 patients.

3. Tympanomastoid segment of nerve is commonest site of lesion.

4. Electrodiagnostic tests are of great help regarding the prognosis of facial paralysis.

5. Steroids or vasodilators have no benefit over a placebo in treatment of Bell' palsy. Morethan 90 percent patients recover spontaneously.

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Summer Diarrhoea of 1994 in Children : New Cholera Strains

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ABSTRACT

1593 cases of diarrhoea were seen between the month of August and September 1994. Out of these 116 were with Ricewatery stools and shock. Culture for Cholera was conducted in thirty. Out of these seventeen were positive for Cholra-Vibrio Serotype Ogawa, V.Cholra Ol, V Cholera 0139. Five out of these died and the rest responded to the Supportive thereapy with I.V Chlromphenical.

RESULTS AND REVIEW

As summer progressed there was steady rise in cases of Gastroenteritis. In the month of June 1994, Seventy four (74) cases of gastroenteritis were admitted, who were discharged after giving intra-venous fluids and drugs. No case succumbed. In the month of July 1994, Two hundred and sixty four cases were admitted with similar complaints i.e, loose motion, watery, at times with mucus or blood, such cases were treated with conventional drugs and were discharged after treatment, no case succumbed. However in the month of August 1994, there was change in clinical presentation. We also received cases with history of rice water like stools and in shock. First such case was an agricultural labourer from Orissa working at Lasjan, subsequently cases stared pouring in bulk with similar clinical presentation from Pulwama, Shopain, Sepden Galwanpora, Pattan, Bijbihara Soibugh belt etc. In total one thousand five hundred ninety three (1593) cases of acute gestroenteritis with moderate to severe dehydration were admitted from Ist day of August to 30th day of September 1994, out of them one hundred sixteen (116) had clinical presentation of rice water stool and shock. Majority of these cases were from District Budgam followed by District Pulwama with male/female ratio of 2;1 and age group in majority of cases was 5-10 years, five deaths occured.

Since this clinical presentation was suggestive of Cholera, microbiology department was approached, which provided some vials for culture (Alkaline Peptone medium). Initially it was reported by microbiology department that no Vibrio Cholera was grown, However, salmonella typhi and pseudomonas were isolated. We treated such cases above the age of two years with intravenous Chlromphenical (70mg/kg body wt.) and fluids calculated as per scoring system and results were encouraging, given the enormous admission, low mortality and followup. At the time of discharge it was being impressed upon the concerned to take boiled water, O R S and other relevent hygeinic measures were explained to them.

Subsequently a team from National Institute of Communicable diseases, New Delhi visited children hospital srinagar on 14th september 1994. Besides having fruitful discussion, they were also apprised about protocol prescribed by us and available results. Stool samples on index were carried by them for evaluation at Delhi and they also provided vials containing transport medium for evaluation here at Microbiology deptt.

Vibrio Cholera Serotype Ogawa was reported to have been isolated by microbiology deptt., of Medical College Srinagar. However drug sensitivity was not mentioned. In another communique from Assistant Director N.I.C.D Delhi, it was reported that V.Cholera 01,+V- cholera 0139 was isolated from the specimens taken. All the vibrio Cholera 01 were sensitive to tetra cycline, Gentamycin and Nalidixic acid, V.Cholera 0139 were resistent to Cotrimexazole, furazolidine and streptomycine. Sensitivity to Chlromphencol was not tested.

Retrospectively on scruitiny, it was found that cases in which vibrio Cholera was reported by NICD Delhi and Medical College Srinagar had also received same regimen and thus it can be recommended with fair degree of confidence for management of such or similar cases. Moreover cost of treatment is also meagre as Chlromphenicol is a hospital supplied drug and popularly used tetracyclines for management of such cases in adults does not find a favour in children.

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Acknowledgement; We are thankful to Dr.M.A.Shawl Medical

Superintentant and other staff of Children hospital for cooperation.

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Burned out Testicular Tumour

A. R. Khan,* M. D. Nasser-u-din,** MBBS.

INTRODUCTION:

Authors wish to report a rare but interesting case of burned Out Testicular Tumour, with widesperead dessimination of embroyonal carcinoma. Primary testicular tumour was represented by enlarged testis, replaced by acellular fibrous scar. Exact underlying biological regression of primary tumour is not clear. However, the lesson is that the diagnosis of primary extra germ cell tumour is not valid, until and unless gonads are ruled out as primary site. Value of fine needle aspiration biopsy cannot be over-emphasied particularly in poor risk patients or prevailing customs, when laparotomy or necropsy cannot be contemplated.

CASE HISTORY

A 45 year male (A.R.) presented with severe backache and pain upper abdomen 3 months preceeded by painless testicular swelling 9 months. Patient also complained of severe generalized weakness and loss of weight. Physical examination revealed a sick person, mildly anaemic with hard mass in upper abdomen and non-tender right testicular enlargements.

LAORATORY FINDINGS:

Hb. 11.8 gm%, with ESR (80 mm/Ist. hour westergen's method) and para-aortic abdominl lymph node mass on ultrasound. X-ray chest was clear. Right Orchidectomy was done and the specimen sent for histology.

PATHOLOGY FINDINGS:

Testis enlarged with intact tunica albuginea C/S revealed entire testis replaced by a rather fibroid like tumour and measuring 58x30x30 mm.

Microscopy showed entire testis-replaced by acellular dense fibrous scar. No residual tumpur seen. Due to inavailability of history of abdominal hard mass, diagnosis of benign fibroma like lesion was made. But on availability of the history of para-aortic hard mass in abdomen, the impression of the burned out testicular germ cell tumour was conveyed to the surgeon. Fine needle aspiration of abdominal mass in the afternoon of the same day confirmed the diagnosis of metastatic embryonal carcinoma in para-aortic lymph node mass. (Fig-I)

Patient deteoarated fast in spite of chemortheraphy and died with generalized metastasis after 4 months.

DISCUSSION:

Pym is credited with the first report of tsticular scar and brown pigment associated with choriocarcinoma metastasis. Subsequent reports include among others those of Heaney's Roth, who analysed 17 cases of realted tumours from the files of testicular tumour registry of the armed forces institute of Pathology - which included Chorio-carcinoma 8 cases; Embryonal carcinoma 5 cases; Mixed embryonal - choriocarcinoma 3 cases and terato-carçinoma 1 case(1). Patient were all young men and included all those subjected to necropsy. Excluded were all cases with more than 5 mm viable tumour in testis. A number of controls were also studied. These included 80 cases of gonadal mesenchymal testicular tumours, 65 cases of accident deaths and 25 cases of primary mediasterial teratiod tumours. The findings of the burnt out testicular tumours are summarized as under[2,3,4]:-

1. Acellular collagen scar in 2 cases
2. Scar with siderophages 7 cases
3. Scars associated with hematoxylin staining bodies 13 cases (Hematoxyling bodies composed mainly of DNA in combination Mixture of phospholipid, proteins scantry 1-2 glycol group and even calcium in some cases).
4. Benign teratomatous elements in 8 cases.
5. Microscopic foci (less than a fraction of high power field) of seminoma in a few cases.
6. Atypical cells filling the tubules adjacent to or nearby scar area in some cases. (This corresponds to in the situ Germ Cell tumour of SKAKEBACK).
7. Testicular atrophy with atrophic tubules hyalinisation of basement membrance and leydig cell hyperplasia.

In the present case it was not easy to make diagnosis from any testicular specimen alone. No hematoxylin bodies or viable germ cell tumour of any kind were seen. Acellular collagens connective tissue was replacing the entire testicular mass (Burnt out testicular germ cell tumour). However, the forthcoming history of hard para-aortic mass followed by fine needle aspiration, with diagnostic smears of embryonal carcinoma (Figure-1) clinched this diagnosis. Refusal of the patient to accent full multiagent chemotherapy was followed by rapid deterioration and generalised metastasis to lung, mediastinium and brain. Patient died after a period of 4 months.

Professor of pathology **Postgraduate Resident Goverment Medical college, Srinagar.

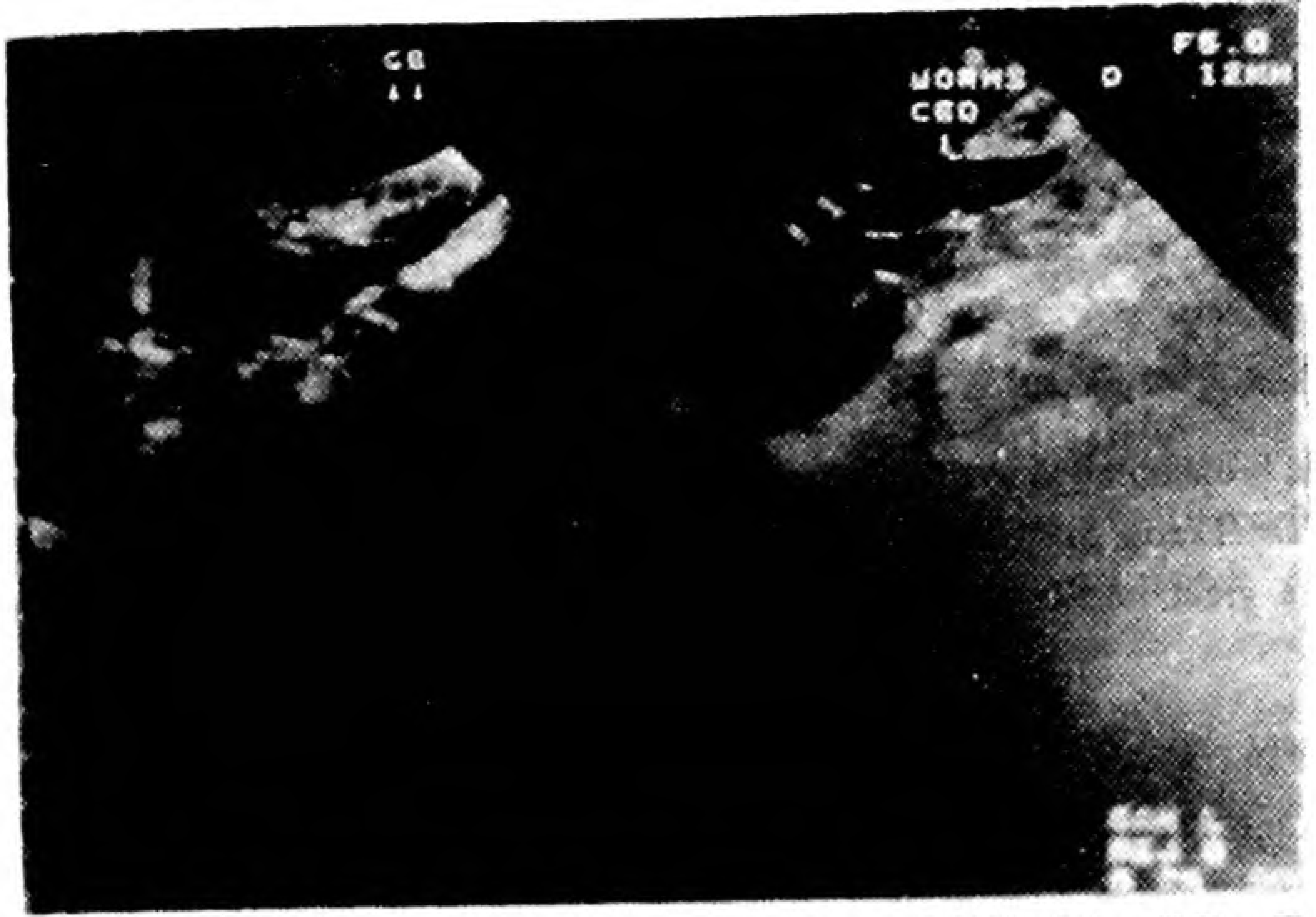

Fig. : FNAC of periaoratic mass shows embyonal carcinoma. (Giemsa * 400)

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The JK - PRACTITIONER Journal will henceforth reccive and consider for publication, manuscript prepared in the Vancouver Style (see Lancet : Feb, 24, Page 429, 1979)

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(a) Title Page
(b) Abstract and Key Words
(c) Text
(d) Acknowledgments
(e) References

Tables (Each table complete with title and foot notes on a separate page) Legends for illustrations

Illustrations must be good quality, unmounted glossy prints no larger than 14.4 by 9.5 c.m.

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Type the manuscript with margins of atleast 2.5 cm. Double spacing should be used throughout, including title page abstract, text, acknowledgment, reference, tables and legends for illustrations. Following sections should begin on seperate pages : Title Page, abstract and Keywords, text, acknowledgments, references, individual tables, and legends. Type the page number in the upper right-hand corner of each page beginning with title page.

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The title page should contain (1) a concise and informative title (2) a short running head of footling placed at the foot of the title page and indentified; (3) first middle intial and last name of each author with academic degrees(s); (4) name of the departments (s) and institution(s) to which the work should be attributed; (5) name and address of author responsible for correspondence about the manuscripts; (6) name and address of author to whom requests for reprints should be addressed.

Fibreoptic Bronchoscopy

G.Q. Khan MD., FCCP
"Associate Prof. & Head Bronchoscopy section GMC Srinagar."

The flexible fibreoptic bronchoscope was first introduced to clinical use in 1967 by Dr. Tkeda. Since then the fibreoptic bronchoscope has made a great impact on the practice of respiratory medicine.

Procedure: The patient should be explained about the procedure, followed by a written consent from the patient.

Premedication: If premidication is to be used, atropine (0.6-1.2mg) injected instramuscularly 30-40 minutes before the procedure. Short acting benzodiazepins (for example midazolam) releive anxiety and provide a degree of amnesia for the procedure.

Positioning of patient: Most bronchoscopists prefer to face the semi-recumbant or sitting position throughout the procedure, others stand at the head of the supine patient.

Topical anaesthesia: Lignocaine (2-4%)is considered safer and is most widely used local anaesthetic agent. It can be used as a spray from a hand atomiser or pressurised aerosol container or by introducing lignocaine gel. A transtracheal injection of about 5ml of 2% or 4% lignocaine through the cricothyroid membrane is increasingly used to anaesthetise the tracheobronchial tree.

Insertion(Pernasal):- Having waited for effective local anaesthesia, the tip of the fibroptic bronchoscope is passed through the chosen nostril. The shaft can be lubricated with lignocaine gel. The largest passage is the inferior meatus, below the inferior turbinate along the floor of the nose. This route should be followed by passing the fibroscope directly backwards not obliguely upwards. As the back of the nose is reached, the fibroscope should be angled much more caudally (about 60°-80°) to pass through the nasopharynx into the space behind the tongue.

Peroral insertion:- When using the oral route either the sitting or supine posture may be used. Back of the tongue should be anaesthetised first. An annular bite guard is threaded over the fibroscope and held in readiness. The patient now opens his mouth and protudes his tongue. The fibroscope is introduced as far as the back of the tongue and the tip is then deflected caudally by 50°-60°.

Insertion through larynx:- Once larynx is in view vocal cords should be inspected during phonation and the movements of the cord noted. During the quite inspiration the fibroscope is passed slowly and very gently through the glottis and the cords are negotiated until the fibroscope reaches the trachea. The trachea, right upper, middle and lower lobes and segmental bronchi and then left main bronchus and left upper and left lower lobe segments are seen in succession.

Disadvantages: The advantages of the fibreoptic bronchoscopy are many and obvious, but the potential disadvantages and possible complications of fibroscope should be carefully considered. These disadvantages are as follows.

1. The view obtained is easily obscured by the secretions, which may be removed by irrigation via sucking channel or wiping the tip on the bronchial mucosa, but sometimes removal for clearing is necessary and repeated reinsertion can be tedious and uncomfortable for the patient.

2. The rigidity of the bronchial tree cannot be felt by fibreoptic bronchoscope, this can be sensed easily with a rigid tube or forceps.

3. Foreign body removal is limited.

4. Control of significant haemorrhage, caused by instrumentation may prove impossible. Vision is rapidly obscured and sucking facilities are limited. In the bronchitic patients blood gases can be deranged simply by passing the fibrescope alone. But these limitations are only relative and minor.

Indications for bronchoscopy:

1. Haemoptysis:

This is a major and most common indication for doing bronchoscopy. profuse or repeated haemoptysis whether with or without radiological abnormalities or physical signs should always lead to bronchoscopy.

2. Coughing, wheeze, stridor and dyspnea:

Cough of recent onset unexplained and persistant with or without sputum must always raise the possibility of a bronchial lesion, a foreign body or bronchial distortion. Change in cough habit may be the first presentation in a bronchial carcinoma.

3. Diffuse pulmonary shadowing:

Bronchoscopy and transbronchial pulmonary biopsy may be indicated in cases of diffuse pulmonary shadowing.

Bronchial biopsy may also give the diagnosis in the cases of miliary tuberculosis, lymphangitis, carcinomatosis, and sarcoidosis.

4. Aspiration:

The possibility of an aspirated foreign body particularly in children must newer be forgotten when taking the history of a cough of recent onset associated with chocking or holding objects in the mouth. Bronchoscopy is urgent on suspicion.

5. Bronchial Obstruction:

Certain syndromes, like pulmonary collapse, either segmental, lobar or whole lung, obstructive overinflation, repeated infections in the same area of the lung and pheumonia that does not clear satisfactorily with antimicrobial treatment, need bronchoscopy for diagnosis.

6. Bronchoalveolar lavage:

This is the sampling of cellular and acellular contents of the epithlial lining fluid of the lower respiratory tract. After insertion the bronchoscope is wedged in the fourth or fifth branching commonly in the right middle lobe. 20ml normal saline are instilled till 100-150 ml is installed. The fluid is then withdrawn by gentle suction in a trap. The fluid is then analysed for cellular and acellular contents. Normally about 40-60% of fluid is recovered. Normally the alveolar macrophages make about 92%, lymphocutes 7% and neutrophils, mast. Cells, eosinophils and basophils less than 1% of the cells seen. broncholaveolar lavage may be diagnostic in certain diseases.

Complications: The fibreoptic bronchoscopy is a safe procedure, however, some complications do occur occassionaly. There can be *tachycardia* by about 43% in some patients. *Fever* may occur in about 2.7% of patients. The major complications like *bleeding* following biopsy or brush use, *pneumothorx* after transbronchial biopsy is seen in 1 to 43% of patients. *Hypoxaemia* has been reported especially in those with respiratory compromise.

Recent advances: Video bronchoscopy is available. Ultrasonic probes to evaluate submucosal tumours prior to surgery are being developed.

Abstract and key words

The sceond page should carry an abstract of not more than 150 words stating the purpose of the study or investigation, basic procedures, main findings (state specific data and their statistical signidicance, if possible), and the conclusions in principal.

Text

The text of observational and experimental articles is usually-but not neccessarily-divided in to section with the headings, introduction. Methods, Results and Dicussion. To clarify their content, especially the results and dicussions sections long articles may need subheadings with some sections.

Acknowledgments

The persons who have made substantive contributions to the study can only be acknowledge. It is author's responsibility to obtain written premission from the persons whose name have been acknowledged because readers will infer their endorsement of the data and conclusion.

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Type each table on a seperate sheet using double space. Supply a brief title for each table numbering consecutively. Give each column short of abbrevitated hesdings. All nonstandards abbreviations that are used ineach table should be explained in footnotes. For footnotes, use the folowing symbols in this sequence : * , +, +, $, II, 88< ++, ... Identify statistical measures of variations such as SD and SEM. Omit internal horizontal and vertical rules. Cite each table in the text in consecutive order. Obtain permission and acknowledge fully, if you use data from another published or unpublished source.

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Three complete sets of figures should be submitted which are professionally drawn and photographed. Frechand or type written lettering is unacceptable. Send Sharp, glossy black and white photographic prints, no large than 1.44 and 9.5 cm. Letters, numbers, and symbols should be clear and even throughtout, and sufficient size that when reduce for publication each item will still be legible.

Each figure should have a label pasted on its back indicating the number of the figure, the name of the authors, and the top of the figure. Do not write on the back of the figures or mount them on cardboard, or scratch or mar them using paper clips. Do not bend figures.

A new self-expandable, nickel-titanium coil stent for esophageal obstruction

"Tamar S, Mordechay B, Oren G., Eran G., Illie C., Dov W., Alex F."

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Carcinoma of the esophagus and gastric cardia causes progressive dysphagia. More than half of these tumors are not amenable to surgical treatment, and even after resection about 20% of these patients will have further dysphagia caused by either recurrence or anastomotic stricture. In about 25% of cases radiation therapy of the tumors is followed by dysphagia.[4] Most of patients are severely malnourished, and dysphagia causes their quality of life to deteriorate. Palliative treatments by means of repeated dilations, Nd:YAG laser photocoagulation therapy, and insertion of plastic esophageal prosthesis are of limited effectiveness and have high rates of morbidity and mortality. Self-expandable metal stents have been described for bronchopulmonary, urinary, and vascular stenoses. Recently, such stents have been used in biliary tract strictures, showing a decrease in the rate of reobstruction and cholangitis when compared with plastic stents. Expandable metal prostheses have also been utilized for malignant esophageal stenoses, and palliation of this nature seems to be more effective than other modalities. However, the main problems with these metal stents are tumor ingrowth leading to reobstruction, migration of the stent from its original position, and epithelial trauma by the distal, hard edges of the stent. The new metallic, coil stent used by our group was developed to solve the above mentioned problems. We report our experience in patients with malignant esophageal obstruction palliated with the new stent.

Stents and placement technique :

The prosthesis we use is a coil spring, made of flat wire, super-elastic nickel titanium alloy . This coil stent is wrapped tightly on an introducing catheter with a wire holding its distal and proximal ends to a total external diameter of 9 mm. A central channel in the delivery catheter permits the passage of a regular guide wire of 0.035 inches. The stent is placed under fluoroscopy with the use of conscious sedation. After endoscopic insertion of a guide wire through the stricture, the introducing catheter and the stent are pushed over the guide into the stricture. The stent is then released, by pulling back the holding wire, to expand spontaneously within several minutes to its original diameter of 15 to 18 mm and length of 8 to 10 cm (Fig.1). The introducing catheter and the guide wire are retrieved after the stent is released and expanded. With the exception of the first stent, the others were built with wider ends (4 mm more than mean diameter)to prevent migration. Endoscopic examination was performed immediately after insertion and repeated 4 weeks later or as needed thereafter.

CONCLUSIONS

This stent is unique in that, it is a simple coil,with close loops made from a single flat wire of nickel titanium alloy. Because the radial force of this material is much stronger than stainless steel, expansion time is therefore faster and the stent is able to dilate even extremely resistant strictures. Pre-stent dilation of about 10 mm is necessary only in very tight strictures.

RESULTS

- Significant and sustained alleviation of dysphagia-in all 36 patients.
- Procedure duration (mean) 10 minutes.
- Time for full time expansion: (mean) 5 minutes.
- Median survival : 17 weeks (range 1-51 weeks).
- Early complications: none.
- Late complications: migration to the stomach, breakage, clogging-six patients.
- Replacement: two patients.

Technical Characteristics of the New Stent (InStent Inc.)

STENT'S CHARACTERISTICS

- Metallic self expandable coil.
- High radial force and wide diameter.
- Flexibility, easy insertion and possibility of removal.
- Tight coiling to prevent tumor ingrowth.
- Safety.

2/2 No. 3.

CLINICAL IMAGES

A 37 years femal with History of Recurrent Biliary Colics but normal Exam. as well as LFT.

Fig. I Ultrasound Liver Shows Hypoechoic longitudnal lesions in a dilated CBD (Bilary Ascariasis)

Diagnosis : Biliary Ascariasis.

Courtesy : G.M. Malik, B.A. Lone GIT Division G.M.C. Srinagar Kashmir-190009.

Alopecia Areata

Qazi Maqsood (MD)
"Asst. Prof. Department of Dermatology & Leprosy G. M. C. Sgr."

Alopecia Areata (AA) is one of common dermatological problems faced in clinical practice. About 1-2% of population is effected. Age is no bar, however very few patients are seen below age of 5 years. In 70-80% cases first attack is seen between 5 to 40 years. Recurrences are a common feature and there is no predominance of any sex. Exact aetiology is not known, various factors implicated in aetiology.

(i) **Genetic factors:** A positive family history is obtained in 10-20% of cases. Identical twins may be affected simultaneously and at the same site.

(ii) **Immunological factors:** It is widely agreed that A.A. is an autoimmune disease, although evidence at present is indirect. It is suggested that in herited defect may involve an increased propensity to form certain organ specific antibodies. Increased association of A.A. is seen with other immunological diseases like Thyroiditis, diabetes, vitiligo and pernicious anaemia. Strongest direct evidence of autoimmunity is constant presence of lymphocytic infiltration in and around hair follicle.

It is suggested that antibodies are formed against matrix cells of hair follicles. Response to steroids both local and oral which are immuno-supressive and to Dinitrochlorobenzene which stimulates cell mediated immunity also point towards immunological cause.

(iii) Pregnancy: Pregnancy has a beneficial affect on A.A. possibly due to higher steroid levels during pregnancy.

(iv) **Psychological factors:** Some investigators consider emotional factors to play a significant role. Recurrences during psychological stress are a common observation however the disease is primarily not a psychosomatic disease.

Clinical features:

The onset is insidious and the first patch may be noted by a relative, parents, hair dresser or suddenly by the patient himself. The patch is oval or rounded in shape with regular margins. It is totally devoid of hair and in an activety spreading lesion or on early stages exclamation mark hair are seen at margins. Most of these lesions heal of their own. In some cases fresh lesions start appearing 2-6 weeks after the primary patch. In rare case the onset may be acute and scalp, beard and moustache hair may be lost in few weeks time. In children typical patch may not be seen, it may be irregylar and sometimes may appear on occiput and spread like a band around scalp.

Clinical variants:

(i) Alopecia totalis: Loss of all scalp hair is called alopecia totalis. The onset may be acute or it may develop within months of partial alopecia, usually within 2 years. 5-10% of cases of partial alopecia progress to Alopecia totalis and it is common in children.

(ii) **Alopecia universalis :** Loss of all scalp and body hair is called alopecia universalis. Progress may be acute. In cases with partial alopecia progress is slow.

(iii) **Loss of hair from other sites:** Other than scalp is seen in 10% cases. It may involve eyebrows, eyelashes, beard, public and body hair. These sites may be involved singly or in combination.

(iv) **Loss of pigmemted hair:** Pigmented hair are lost quicker and a person may appear to grow "white overnight". This is especially in patients with grey hair and in those with underlying vitiligo.

Prognosis:

No reliable opinion can be given at any stage. Most of the cases heal of their own. If progress is slow prognosis is good and if progress is acute, prognosis is not that good. Relapses are more common with acute cases.

Treatment

The course is unpredictable. Response to treatment is good, however relapses are common. Various treatments employed are:-

(i) **Reassurance and Tranquilisers**

(ii) **Mild irritants** like Tincture of iodine, Resorcinol, Dithranol tar etc. are employed in most cases.

(iii) **U.V.L.:** exposure to UVL has been reported to be helpful.

(iv) **Photochemotherapy:**

  (a) Local application of psoralens and controlled exposure to sunlight or ultraviolet light.

  (b) oral psoralens and exposure to ultraviolet or sunlight is also em ployed.

(v) **Minoxidil:** Topical application of 2% minoxidil is advised on twice a day schedule.

(vi) **Trichosacharide:** "A mucopolys-accharide extract of natural mammalian tissue" is being advocated for treatment.

(vii) **Placental extract:** Both topical and intralesional have been used with varriable results.

(viii) **Dinitrochlorobenzene:** Use is in experimental stages.

1992, 4 : 2568

Photodermatol 1985 ; 12 : 78-84.

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Kashmir (INDIA)

Dr. G.M. Malik, M.D.,FACS

Learn To Stop Aids

"IS IT TIME TO TALK ABOUT AIDS IN INDIA?"

It has entered the country virus which causes Aids is beginning to spread, aids by ignorance and fear. Understanding aids is the way to keep it away. It remains hidden in the person for years before manifesting itself as aids. It may quielty enter our lives. Be cautious. Aids is incurable.

What is Aids?

A Acquired
I Immuno
D Deficiency
S Syndrome

A disease caused by a virus: Human immuno deficiency virus (HIV) which destroys the cell of the body which fight against diseases thus making the person vulnerable to infections and certain cancer which healthy bodies (Persons) can resist.

1. Sexual intercourse with an infected Partner.
2. Use of unsterilized needles or syringes infected with the Aids virus (HIV), especially drug users.
3. An infected mother may apss iton to her child. The chance is about one in three(OR 30%).
4. Transfusion of blood infected with the virus (HIV).

Ways of Prevention

Condoms are effective in preventing Aids and other sexually transmitted diseases (STDs) like syphilis and gonorrhoea.

Multi-Sex partner increases the risk of contracting Aids and other STDs.

In case of a STD, immediate and adequate treatment should be sought.

STDs (Gonorrhoea, Syphilis and others) faciliate the transmission of the Aids virus (HIV).

See that the needle and syringes are sterilized (eample-boiling for 25 minute before use) Sterilizeskin piercing instruments, acupuncture needles, etc.) before reusing aviod taking drugs. Never share needles.

See that the blood or blood products to be transfused are tested for the aids virus (HIV).

In an emergency or druging lack of test facilities avoid blood from unknown doners. Limit blood transfusions for life saving only.

1. SHAKING HANDS
2. HUGGING and KISSING
3. COUGHING & SNEEZING
4. PUBLIC TOILETS
5. SHARING MEALS, UTENSILS & DRINKS
6. SWIMMING POOLS
7. MOSQUITO INSECT BITES
8. SHARING CLOTHES

IEARN TO USE A CONDOM THE CORRECT WAY

1. Hold the top of the condom. Press out the air from the tip and roll the condom on, using both hands.
2. Roll the condom right to the base of penis leave space at the tip of the condom for the semen.
3. Afterjaculation , hold the condom at the base and slide it carefully off.
4. Makeaknot and dispose off the condom in a pitlatrine

Salient facts

Almost all people infected with HIV will eventually develop aids.

There is no vaccine to prevent HIV infection.

People having HIV infection may not show manifestation of aids for many years. Thus they will be unaware and may spread the virus unknowingly.

There is no treatment for aids

The drugs available only increase life span by a few months and are very expensive.

There is no risk in donating blood.

A mother with the aids virus should continue to breastfeed her baby.

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EDITORIALS:

"FIGHT AGAINST CANCER"

Cancer can arise in any organ or tissue of the body. It is not one disease but rather a collection of more than 100 diseases. The variation being dependent on the involvement of site and histopathological type.

We know the cause or at least risk factors in some cancers and can prevent their occurrence. Cancer is therefore, to some extent avoidable. Environmental factors well known in etiology of some cancers are not only man made, but also include other factors like tobacco, diet and other natural carcinogens. When a known risk factor is eliminated, certain cancer incidence goes down as noted in decreased prevalence of cancer cervix in the West. Epidemiological surveys show that many tumors such as cancer cervix, oral cavity, esophagus are more frequent in developing countries whereas certain cancers like lung and breast are more prevalent in developed countries. The development of preventive measures specific to preventable cancers could lead to a significant fall in the incidence of these cancers. However the lack of knowledge of the intimate mechanisms by which the cancer is produced stands in the ways to prevent it. Attempts should be made to control cancer by "Secondary prevention" that is by detection of cancer in early stages. It is achieved by screening high risk population, for most commonly occurring cancers. In few countries, screening programmes are launched for breast cancer, cancer cervix and endometrium in women are example in Secondary prevention in Japan. We know, all over the world outcome of cancer stomach is very poor with 5 year survival as low as 5-10% only. But in Japan, study is totally different. The Japanese have demonstrated the value of mass surveys for gastric carcinoma in their population. They have detected cases in early stages by the use of mass upper gastrointestinal surveys. Surgery is highly successful when gastric cancer is limited to mucosa but incidence of such early cases is less than 5% in most of the countries including West. However in Japan, the incidence of lesions confined to mucosa or submucosa was only 3.3% in 1955 but because of screening procedures, this has increased considerably with corresponding survival rate of 90%, in other words, through secondary prevention cancer stomach at least in Japan has become curable cancer which is still incurable in rest of the world. We should follow Japanese guidelines at least in Kashmir valley as cancer esophagus and stomach are the commonest cancers encountered in our valley. In addition attempts should be made to identify cancer causing agents or risk factors involved in the initiation of cancer so that prevention strategies can be built on this information, then only we can motivate people to avoid risk factors.

It has been demonstrated that the optimal use of therapies as surgery, Chemotherapy, Radiotherapy, and Hormone therapy can cure one out of three cancer Patients at least in developed Countries and with modern multidisciplinary treatment, the cure rate is much higher than is often thought of by public. The mass media should be used to educate the public for early diagnosis.

Seven Warning Signals of Cancer are:-

1. Change in Bowel or Bladder habits.
2. A sore that does not heal.
3. Unusual bleeding or discharge.
4. Thickening or lump in breast or elsewhere.
5. Indigestion or difficulty in swallowing.
6. Obvious change in wart or mole.
7. Nagging cough or hoarseness.

Knowledge available today allows us to predict that given the right measures, sufficient resources and continued goal directed research upto one third of existing cancer can be prevented, upto one third can be cured and most incurable cancer patients can be spared pain.

Lastly, death in cancer is often a prolonged procedure and is often associated with pain. Efficient, in-expensive pain relief measures are available for such cancer patients. Death is inevitable for all of us but death in pain is avoidable.

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GLOBAL TUBERCULOSIS INCIDENCE DURING 1990-2000

In 1990 WHO routinely collected data on the number of tuberculosis cases in Member State each year. Within each WHO region, except the African region, an overall regional crude incidence rate was calculated by estimating the incidence in the most populated countries. Notification data were considered reliable when provided by programmes with an established surveillance system. Reliable notification data were preferentially used for these estimates. For countries with unreliable notification data, the annual risk of infection was used to estimate incidence.

In estimating future incidence, allowances were made for demographic factors (changes in the size and age structure of regional population) and epidemiological factors (changes in underlying incidence rates). To accurately allow for both demographic and epidemiological factors, regional age-specific incidence rates for the years 1995 and 2000 were estimated and then applied to regional age-specific population projections for 1995 and 2000.

This was undertaken in two steps. First, data available at WHO on the age distribution of notified cases in each region during 1990 were applied to the 1990 regional crude incidence rates to derive the 1990 regional age-specific incidence rates. Second, trends in regional notification rates during 1985-90 were applied to the 1990 region are specific incidence rates to derive estimates or regional age specific incidence rates for the years 1995 and 2000. This assumes that further age specific trends will remain unchanged. These rates were then applied to regional age specific population projections to calculate the number of incident cases expected in 1995 to 2000.

Current data suggest that between 5% and 10% of persons co-infected with HIV and Mycobacterium tuberculosis will develop tuberculosis each year, compared with less than 0.2% of persons infected with M. tuberculosis but no HIV. From these data, we estimate that around 95% of HIV infected tuberculosis cases are attributable to HIV infection (i.e. attributable risk = 95%) and the remaining 5% of co-infected cases would have developed tuberculosis regardless of their HIV status. This attributable risk was applied to estimates of HIV sero-prevalence among patients with tuberculosis to derive the number of HIV attribute tuberculosis cases (i.e population attributable risk) in 1995 and 2000. The number of tuberculosis deaths attributed to HIV infection were estimated by applying the regional case fatality rates to the estimated number of HIV attributable cases.

It is estimated that there were 7,537,000 active cases of tuberculosis in 1990. Over 4-5 million cases (65%) occurred in the South East Asian and Western Pacific regions, including 2.1 million of India, 1.3 million in China, and 0.4 million in Indonesia. One million cases are estimated to have occurred in Sub Saharan Africa, 0.2 million cases in Eastern Europe and independent states of the former USSR, and 0.2 million cases in Western Europe and other industrialized countries.

Global incidence is predicted to increase from 7.5 million new cases annually in 1990 to 10.2 million new cases by the year of 2000, and increase of 36%. Over 3.9 million cases annually are expected in South-East Asia, 2.3 million in the Western Pacific, and 2.1 million in Sub Sharan Africa by 2000.

For South East Asia and Central and South America, age specific and crude incidence rates are expected to fall during 1990-2000. However, the rate of decline is slower than in previous years because of the expected spread of the HIV epidemic. The actual number of new cases will continue to increase because of population growth.

In Eastern Europe and states of the former USSR, notification fell during 1985-90. However, it is not clear that this decline in incidenc rates can be maintained under current soco conditions in these countries. It is predicted that tuberculosis incidence rates in Eastern Europe and states of the former USSR will remain a their 1990 level in the near future. Notification rates for some Western European and other industrialized countries (e.g. Netherlands Norway and USA) have recently increased after years of decline rates in other Western European industrialized countries may also increase in the near future, partly because of increasing migration of people from regions of the world with a higher incidence of tuberculosis, and partly because of the increasing number of persons with dual HIV and M. Tuberculosis infections. It is estimated that tuberculosis incidence rates in Western Europe and other industrialized countries will either remain at there current levels or increase slightly during the decade.

It is estimated that 0.3 million of the 7.5 million new tuberculosis cases in the 1990 were attributable to HIV infection.

By the year 2000, 1.4 million (14%) of the 10.2 million new cases acquring each year will be attributable to HIV infection.

Some 2,530,000 tuberculosis deaths acqured in 1990, including 1.1. million in the South-East Asia region and 0.6 million in the Western Pacific region. Globally, 116,000 tuberculosis deaths (0.4%) were attributable to HIV infection in 1990, most of which occurred in Sub-sharan Africa.

Nearly 90 million new tuberculosis cases and 30 million tuberculosis deaths are expected to occur during the present decade with the present level of interventions. For a disease where interventions are known to be cost-effective, this is truly staggering.

Current intervention strategies are expected to result in substantial reductions in age-specific incidence rates in the Eastern Mediterranean region and central and south America, and to a lesser degree in the Western Pacific and South-East Asia regions. However, the total number of new cases in these regions is predicted to increase in the near future because of population growth.

It has been demonstrated that effective application of short-course chemotherapy in well managed national tuberculosis programmes produced excellent results, even under the most adverse conditions. Short-term chemotherapy of smear-positive tuberculosis cases in one of the most cost-effective health interventions available. A higher priority must be given to this disease, both by the countries most severely affected and by the donor countries which invest in health care programmes in these countries.

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DIAGNOSIS OF BUDD-CHIARI SYNDROME-

DO WE NEED ANGIOGRAPHY?

The obstruction to hepatic venous outflow tract may occur at the level of sublobular hepatic veins (veno-occlusive disease), major hepatic (HV) (Budd Chiari Syndrome) or at the level of suprahepatic inferior vena cava (IVC) prior to its entry into right atrium. Budd-chiari syndrome is however loosely used to refer to all cases of hepatic venous outflow obstruction (HVOO). The diagnosis of HVOO is traditionally made by liver biopsy. The site of block in hepatic venous outflow tract is then confirmed by angiographic studies which are invasive , not easily available, require expertise and are associated with definite, albeit, small morbidity[1]. Recently a number of non-invasive modalities like ultrasonography (US)[2-5],CT scan[6] and MRI[7], have been used for its diagnosis. US is most easily available and safe procedure for this purpose, but its role in diagnosis of site of block (major HV and or IVC) in HVOO has not been systematically evaluated. HVOO is not an infrequent disease in India[8,9]. US evaluation by the gastroenterologists is rapidly becoming a part of clinical work up the patients.

Over a three year period (January 1988 to February 1991) we prospectively evaluated the efficacy of US (Toshiba 3.5 MZ linear scanner) in delineation of the site of block (major HV and or IVC) in 43 consecutive patients of HVOO admitted with us during the said period. The US feature of HVOO included blockage of Hv and or IVC with proximal dilatation and with evidence of intra-hepatic venous (hepatic vein to hepatic vein) collaterals. The diagnosis of HVOO was confirmed in all cases by liver biopsy and angiography. The details of methodology and the preliminary results have already been published by us[10]. Four out of 43 patients of HVOO had patent major HV and IVC on US and angiography and were labelled as cases of Veno-occlusive disease. The results of comparative efficacy of US in delineation of site of block in the remaining 39 patients with block HV and or IVC are shown in Table I.

Overall the site of block in the HV and or IVC was correctly identified in 37 (94.8%) out of 39 cases of HVOO.

With our experience we conclude that except for patients with veno occlusive disease, in which the obstruction lies in the sublobular HV and central sinusoids (which are not discernible on US), all cases of HVOO can be confidently diagnosed by US which is easily available, cheap, safe and highly accurate (94.8% accuracy) in diagnosis of site of block in HVOO. US has additional advantages, of simultaneous diagnosis of (a) intra-hepatic space occupying lesions (which can cause secondary HVOO) (b) etiology of IVC obstruction (tumor, thrombosis etc.) (c) signs of portal hypertension, besides providing the information about size of portal and splenic veins, useful for purposes of therapeutic protosystemic shunts.

Recent use of doppler ultrasound[11] and two dimensional doppler echocardiographic studies[12] have further established the role of US in diagnosis of HVOO. Attempts have also been made to diagnose Veno-occlusive disease with the help of doppler studies[13]. With increasing experience with non-invasive tests we think that the need of angiographic studies, for diagnosis of patients with HVOO, may soon be obviated.

TABLE-I

Site of block	Angiography	US	Sensitivity	Specificity
Hv alone	14	13	93%	100%
IVC block alone	22	21	95%	100%
IVC+HV combined block	03	5	100%	94.45%
Correct diagnosis of site of block	39	·37		

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Corrections

There are a few corrections in JK-Practitioner Vol.2 No.1 Jan-Mar 1995.

Figure on page 31 represents the chapter on page 35.
Figure on page 35 represents the chapter on page 31.

On page 38 read AIDS do not spread by:
1.SHAKING HANDS
2.HUGGING AND KISSING
3.COUGHING AND SNEEZING
4.PUBLIC TOILETS
5.SHARING MEALS,UTINCILES AND DRINKS
6.SWIMMING POOLS
7.MOSQUITO INSECT BITES
8.SHARING CLOTHES

CURRENT TOPICS:

GENE THERAPY & CANCER

Gene therapy is an in-vivo delivery of DNA, its long term maintenance and expression within physiological levels.

Human genome contains 50,000 to 100,000 genes encoded in DNA sequence. There are two copies of genome in every somatic cell. The rational elucidation of clinical patterns of illness on the basis of specific genetic changes, resulting in abnormal structure and function (the molecular disease), e.g., the reciprocal translocation between break-point cluster (bcr) of chromosome 9 and the abelson proto-oncogene (abl) of chromosome 22, with resultant fusion and expression of a novel 8.5 kb mRNA encoding for a 21,000 dalton chimeric protein which is a nonintegeral tyrosine-kinase having a vital role in signal transduction and probable production of a cancer phenotype.

Fragments of DNA, hundreds of kilobases in size, carry all the information for complete and long term expression of mammalian genes irrespective of position effects and resistance to methylation. Such DNA introduced into most of all cells of the body will only be expressed in the tissues specified by the endogenous promoter and will be under physiological control. The DNA fragment is introduced efficiently using cationic lipids, or receptormediated delivery, both of which work in-vivo. Full levels of physiologically controlled expression can be obtained after transfer of intact genes on the fragments of DNA, as has been demonstrated by transfer of yeast artificial chromosomes (YAC,s) into transgenic mice. Maintenance of Unintegrated DNA for several months occurs in tissue such as muscle and lung without any specific mechanism for replication, degradation or nuclear localisation. Long-term maintenance of un-integerated DNA in dividing stem cells requires replication and segregation which can be supplied by mammalian replication-organic or centromere DNA. This would create a mammalian artificial chromosome (mac), a basic powerful tool for gene-therapy. MAC would carry DNA fragments large enough to drive full and controlled expression of the therapeutic gene and would be maintained stably in parallel with the mammalian chromosome in any cell into which it is introduced, without integration into genome. As no viral elements are involved, there is no risk of recombination with wild type virus or immune reactions against viral proteins and as the DNA would not integrate into cell genome, there is no risk of mutagenesis. (1)

Epithelial-derived neoplasms or carcinomas comprise a major proportion of solid tumors, which are relatively refractory to conventional chemotherapy. Selective expression or transduction of toxin genes to carcinomas (molecular therapy) may offer important advantage over conventional treatment. The viral thymidine kinase (tk) genes are a class of toxin genes that act by metabolising ganciclovir or related anti-viral agents, into a toxin. (2,3) Interestingly, the viral tk genes lead not only to the death of the cells that receive the viral-gene, but also direct a poorly characterized 'bystander toxin effect' on the surrounding malignant cells that did not receive the viral gene. Thus (tk gene) toxin gene therapy does not require every cancer cell receiving the foreign gene for a lethal effect. Similarly a secretory leukopoieses inhibitor (SLPI) gene was expressed in breast, lung, oropharyngeal, ovarian and colorectal carcinomas. The tissue specific transcriptional regulatory sequences were isolated and used to construct a chimeric gene in which the SLPI sequences directed Herpes simplex virus thymidine kinase (HSVtk) expression. SLPI_expressing carcinomas were reduced in number by transduction of the SLPI_directed toxin plasmid plus ganciclovir. (4)

The infection by human immunodeficiency virus (HIV) has remained refractory to treatment till date. Associated with AIDS and other immunodeficiency disorders is a wide-spectrum of neoplasms, viz kaposi's sarcoma,lymphocytic lymphomas, hodgkin's disease, squamous cell carcinomas of head and neck, squamous cell carcinomas of oral cavity, cholacogenic tumors and rectalcarcinomas. (5) Molecular interventions have been developed which inhibits productive HIV replication, viz transdominant proteins, RNA decoys and ribozymes and of late, expression of vectors which optimize production and regulate synthesis of transdominant mutant from a Rev improve its antiviral effect.

The combination of strong constitutive enhancer, a Tat activation response (TAR) regulatory element and transdominant Rev confer increased resistance to HIV replication. Gene therapy is recent in the rubrics of fight against cancer. Over 200 patients worldwide have received exogenous functional genetic material with therapeutic intent. The results of these pioneering experiments cannot be expected to be starting, but it reflects the steadfastness to fight this dreaded disease.

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Continued on page.21

CURRENT TOPICS:

CURRENT STATUS OF AORTIC AND MITRAL VALVULOPLASTY

Fayaz A. Shawl, M.D[*]

For the past ten years, the development and wide spread application of balloon angioplasty has made a dramatic contribution in the care of patients with acquired vascular stenosis. Initially, it was used for peripheral vascular and coronary artery stenosis, hypoplastic and stenotic pulmonary arteries, aortic coarctation and venous obstructions but now balloon dilatation has other applications. Since 1984, balloon valvuloplasty has been used for the treatment of aortic and mitral stenosis.[1-3]

Before the development of prosthetic heart valves, dilatation was performed surgically for both aortic and mitral stenosis. For patients with aortic stenosis, the results were suboptimal and were often complicated by calcific embolization, inadequate relief of gradient, and trauma to the valve apparatus.[4] For mitral stenisis, the story has been quite different and mitral commissuriotomy has been performed successfully for the past thirty years, with excellent long-term results in selected patients.[5]

However, until recently, the management of valvular stenosis has remained exclusively in the province of the cardiac surgeon. While the surgical armamentarium has included a variety of open and closed procedures for revising the structure or function of diseased native valves, the mainstay of surgical therapy has consisted of replacement of diseased valves with biological or mechanical prosthesis.

Although surgical valve replacement or repair is commonly performed, it is a major procedure. In some patients, the risks are high enough that the patient, the surgeon, or the attending physician wishes to defer or avoid surgery. Also, no prostheses is perfect; all have advantages and disadvantages.[6] Some valve prostheses have suboptimal hemodynamic characteristics, whereas others deteriorate with time. Finally, the potential for thromboembolic complications remains.

For these reasons, among others, balloon valvuloplasty has attracted considerable interest. In 1982, Kan and his colleagues[7] from the Johns Hopkins University reported the first successful percutaneous balloon valvuloplasty in an eight year old child with isolated congenital stenosis of the pulmonic valve. Based on the favorable experience with pulmonic stenosis, Lababidi and co-workers[8] began performing a similar procedure in young patients with congenital valvular aortic stenosis who had no associated cardiac defects. Following the experience with balloon valvuloplasty in the management of congenital pulmonic and aortic stenosis, Lock and co-workers[2] developed a method for percutaneous balloon mitral commissuriotomy in young adults with acquired (rheumatic) mitral stenosis. With the success of balloon valvuloplasty in the management of mitral stenosis, Cribier and Letac from Rouen, France[1] extended the techniques to dilatation of stenotic aortic valves in patients with advanced calcific disease.

However, before the technique should be accepted or widely used, several questions needed to be addressed: What is the mechanism of dilatation? Is it safe? Is dilatation effective? What are the long term benefits? Some of these questions cannot be fully answered as yet, and considerable further study is required.

MECHANISM OF BALLOON DILATATION

The mechanism of balloon dilatation depends on the anatomic substrate. For mitral stenosis, dilatation yields a result similar to that attained by commissuriotomy with splitting along the lines of the fused, thickened commissures.[9,10] For the aortic valve, the anatomic substrate is more variable. About 50% of older adults undergoing aortic valve replacement have senile calcific degenerative aortic stenosis with calcific deposits that limit cusp mobility, but do not result in commissural fusion. In a minority of older patients, the valve is biscuspid or related to prior inflammation with commissural fusion.[1] Therefore, the mechanism of dilatation varies depending upon the anatomic substrate. If the commissures are fused, the balloon may split the valve along the commissures, but in most cases of calcified degenerative aortic stenosis, the calcific plates are fractured.[12,13]

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IS DILATATION SAFE?

Safety issues depend on the specific procedure performed and the experience of the operators: equally important is the specific patient population on whom the dilatation is attempted. Naturally, procedures performed on inoperable or "high risk" surgical patients carry with them much greater risks than if the same procedure were performed on "good surgical candidates."

Aortic balloon inflation for as long as one to two minutes is surprisingly well tolerated, typically without severe hypotension, loss of consciousness, or severe angina. In a minority of patients there is significant hemodynamic instability with a marked decrease in systemic pressure to less than 70 mm Hg, and this limits the permissable balloon inflation time. Fortunately, such a hypotensive response does not usually occur in patients with normal ventricular contractility, but it is a potential risk in patients with poor left ventricular function. This is in contrast to patients with mitral stenosis in whom the standard inflation of two large balloons is very often associated with marked systemic hypotension and pulmonary hypertension. These effects, however are quickly relieved after balloon deflation.

Some complications associated with valvuloplasty depend in part on the route the catheter takes (transvenous, transeptal, or retrograde arterial) and whether associated peripheral vascular disease is present. In elderly patients with aortic stenosis who have tortuous calcified iliac arteries, passing the large, rather stiff catheters through the arteries is often difficult. For mitral valve dilatation, a modified transeptal procedure is performed which requires that the atrial septum be punctured to allow the catheter to enter the left atrium. Although a small atrial septal defect may remain, it is not resulted in major clinical problems.[14,15]

For patients with mitral stenosis, the potential for thromboembolism from undetected left atrial thrombi or calcific deposits is of most concern, but fortunately this occurs infrequently. Full heparnization, during and after the procedures, minimizes the risk for embolization. The reported incidence of embolization is less than 2%. In order to minimize the risk patients with mitral valve disease should have a pre-operative two-dimensional echocardiogram in an attempt to exclude patients with left atrial thrombi.

Those patients with a history of thromboembolism should have intensive anticoagulation with coumadin prior to mitral valvuloplasty to decrease the potential of thromboembolism. For patients with aortic stenosis, calcific embolization is very rare, being reported in only less than 1%.[16]

Other complications, such as myocardial perforation and arterial laceration have been reported, but are uncommon. In patients with severe congestive cardiac failure and low cardiac output, the potential for death during hospitalization is increased. When experienced operators perform the procedure, the in-hospital mortality has been approximately five percent.[16]

Concern has been raised about the development of valvular insufficiency after valvuloplasty. Currently the procedure is restricted to patients with no more than mild insufficiency. In such patients, it is uncommon for significant valvular insufficiency to develop, although occasionally, a mild worsening may occur. Paradoxically, aortic insufficiency has actually decreased in some patients, particularly if they had a large aortic gradient prior to the procedure.

A final category of complications relates to the arterial injury at the femoral site. The catheters currently used for the arterial approach are large and they have resulted in peripheral vascular injury such as lacerations, large hematomas or pseudoaneurysmso. Surgical repair of these injuries has been required in approximately 10% of the cases. The continued development of smaller catheters should decrease these complications.

IS BALLOON DILATATION EFFECTIVE?

The goals of dilatation vary and depend on the specific patient. In the elderly high risk surgical patient with severe aortic stenosis, congestive cardiac failure, and other major medical problems, dilatation is clearly performed for palliation. After aortic valvuloplasty, the initial results depend on part of the baseline severity of the lesion. If the stenosis is very severe prior to dilatation, significant residual stenosis often remains. If the valve is only moderately stenotic, the results are considerably better.

In the recent French Registry experience, Cribier et al,[17] reported 442 patients in whom there was a reduction in the mean gradient from 78 ± 29 to 37 ± 19 mm Hg and improvement in the aortic valve area from 0.46 ± 0.18 to 0.79 ± 0.33 cm^2. In some patients, such improvement has resulted in major improvement in left ventricular function and excellent relief of clinical symptoms. In a substantial number of patients, although dilatation may be successful, significant residual stenosis remains. If the patient selection criteria were to include younger patients or those with less severe stenosis, the results might be better because in such patients it is possible to achieve an aortic valve area greater than 1.0 cm^2.

In mitral stenosis the results are better than in aortic stenosis and are often comparable to those achieved by surgical commissuriotomy; the valve can often be dilated to a final mitral valve area greater than 1.5 to 2.0 cm^2. This in part depends upon the patient population; in the young patient with a non-calcific pliable valve the result from balloon valvuloplasty will probably be significantly better than in the older patient with a heavily calcific valve and fusion of the subvalvular apparatus.[18, 19]

WHAT ARE THE LONG TERM RESULTS?

Follow-up data after valvuloplasty are limited. In the short term follow-up of patients with stenosis there is usually an improvement in clinical symptoms which occurs even though there may have been only a modest decrease in aortic valve gradient.[16, 17] However, in patients with severely depressed left ventricular function if there is an inadequate reduction of aortic valve gradient at valvuloplasty there appears to be a substantial one year mortality.

The rate of aortic valvular restenosis ranges from approximately 25% to as high as in the majority of patients.[20, 21] In many cases this may be the result of inadequate initial dilation. Recently Cribier et al have demonstrated that when the valve can be dilated to achieve a valve area >1.0 cm^2, the patients have marked clinical improvement, and also less likelihood of restenosis. This type of very superior result is usually accomplished by using a large balloon.

For patients with mitral valve dilatation the results are considerably better.[14,15] In ideal candidates, the short-term restenosis rate is very low, but in patients with more severe disease, particularly with subvalvular involvement, restenosis rates are higher.[22] It is anticipated that in the "ideal candidate" the long term results will be similar to those seen after surgical mitral commissuriotomy.

SELECTION CRITERIA

In mitral stenosis, ideal candidates for valvuloplasty, like ideal candidates for commissuriotomy, are those who have isolated mitral stenosis (or no more than mild mitral insufficiency), have pliable valve leaflets, and have minimal valvular calcification. In such patients, mitral balloon dilatation may be considered the treatment of choice in centers experienced with the technique. In patients with a severely calcified mitral valve the valvuloplasty procedure can be done but the result may be less than ideal and the restenosis rate may be higher.[18,19] Exclusion criteria include significant mitral insufficiency, left atrial thrombus, severe subvalvular chordal fusion, and inexperience with the procedure.

For aortic stenosis, balloon valvuloplasty should be restricted to three groups of patients; 1. Those who are at high risk for surgery by virtue of associated diseases, advanced age, or severe left ventricular dysfunction, 2. Those with severe aortic stenosis who are good surgical candidates, but who decline aortic valve replacement, and 3. those with severe left ventricular dysfunction who are potential candidates for aortic valve replacement but need valvuloplasty in order to improve ventricular function and lower the risk of subsequent valve replacement.

CONCLUSION

Although aortic and mitral valve dilatations rapidly emerging as a new percutaneous technique, the procedure is still investigational. The equipment will require considerable modification to improve the ease, efficacy, and safety of the procedure. In isolated mitral stenosis, valvuloplasty may become the treatment of choice in carefully selected patients.

Valvuloplasty for aortic stenosis is only a palliative procedure but by modifications of the technique and the instrumentation used in the procedure, future results should improve greatly.

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"UPDATE ON MALE ERRECTILE DYSFUNCTION"

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Male errectile dysfunction till recently was attributed to many myths, and wrong concepts partly due to social and cultural reason and mainly due to lack of proper scientific data in this field. Earlier concepts attributed errectile failure due to anxiety-depression syndrome and till 1960's 90% of male impotence was believed to be due to psycological reasons. However, in last two decades due to innovative laboratory and clinical research in haemodynamics, neurophysiology and pharmacology the mechanism of male errection has been more clearly understood (1-4).

Normal male errection involves smooth muscle relaxation of cavernous sinusiodal spaces in corpora cavernosa due to release of neurotransmitters like vaso-active polypeptide (VIP), Nor-ephenephrine, acetyl choline, resulting increased artrial flow in to sinusiodal spaces. Trapping of blood in cavernosal spaces due to compression of venules between the expanding sinusiods and the non-compliant tunica albuginea resulting in reduction in venous outflow of blood thus resulting in tumesence (errection). Detumascence occurs by reversal of this process by recovery of intrinsic smooth muscle tone, arterial smooth muscle contractions thus resulting in reduction and inflow of artrial blood, and facilitation of passage of outflow blood.

Current concepts in pathogenesis have defined psycological, neurological, vascular, harmonal and sinusiodal factors in causation of impotence.

PSYCOGENIC CAUSES:-

Though exact mechanism of psycogenic cause of impotence is not known. Multiple factors like anxiety, depression, guilt, emotional stress etc., can be involved. Further lack of knowledge of physiological changes occuring in sexual activity with age can be another factor in certain cases. Probably sympathetic overactivity and inhibition of neuro transmitters may be the final operating mechanism in this group.

VASCULAR CAUSES:-

Arterial and veinous cause supplying and draining the penis may be the culprit in a given case. Reduced penile blood supply can occur in senile atherosclerosis, pelvic steal syndrome, leridoe's syndrome, pelvic vascular trauma and diabetic vasculopathy are some of the well known causes(5). Insufficient venous occlusion mechanism due to unknown causes or pyrognic disease etc., can also lead to vasculogenic impotence.

NEUROLOGICAL CAUSES:-

Nerve supply to the errective may be affected by lesion in brain, spinal cord or at peripheral nerve supply level. Thus brain tumour, central vascular accidents, parkinson's disease, spinal cord tumours, multiple sclerosis, tabes dorsalis, diabetic neuropathy, pelvic nerve injuries may be causes of impotence from this group(6).

DRUGS:-

Many drugs can cause impotence these include almost all acute hypertensives particularly, beta blockers, cemitidine, anti depressants and anti androgens.

SYSTEMIC DISORDERS:-

Chronic debilitating disease like cirrhosis of liver, COPD, hypertension and malignancy can lead to impotence due to varried factors.

HORMONAL CAUSES:-

Estrogens, and anti-androgen therapy, hypogonadisim bilateral orchidectomy, diabetes mellitus are some of the causes in this group.

MANAGEMENT:-

A proper history and proper physical examination keeping the above mentioned causes in mind is mandatory. Basic laboratory investigations should include urine analysis, Random Blood Sugar (RBS), serum urea, serum creatinine. History of sudden onset sleep dysfunction, normal nocturnal errections, suggest phsycogenic cause. Full errection on recently developed papavarine test also suggests psycogenic cause. However, assay of FSH, LH, prolactin, testostrone levels will be needed in suspected cases of impotence due to hormonal causes. Full neurological examination including latest biothesiometry and evoked potentials may be needed in selected cases. History of peripheral vascular disease, intermittent claudication will provide useful clue in vasculogenic group. Penile, brachial blood pressure index (PBI) may be necessary in selected cases in this group. Functional test of intactness of penile vasculature can also be determined by recently introduced papaverine test, and Duplex ultrasonography. Pelvic angiography may be needed in selected cases. Nocturnal penile tumesence (NPT) can be determined by special apparatus to differentiate between psycogenic and organic cause of impotence. Venogenic impotence can be confirmed with cavernosometry and cavernosography.(5,7).

Generally treatment has to be individualized depending essentially on diagnosis. Psycogenic cause, has to be treated with reassurance psycotherapy, sexual education and possible empirical drug therapy. If arterial insuffiency is proved, revascularization procedures like epigastro-corporial anastamosis may help, venogenic impotence cases can be benifitted by ligation and stripping of dorsal vein of penis. Most of the patients can be initially tried on self injection of vaso-active drugs like papaverine and PGI(8). Vacume errection devices (VED) and penile prosthesis may be considered in selected cases & Empirical therapy with yohimbine or testestrone may help certain cases (9-13). The treatment options must be discussed with the patient and if possible the wife. Ongoing research in the field of errectile dysfunction is likely to improve the present modalities of treatment and possibly develop newer options.

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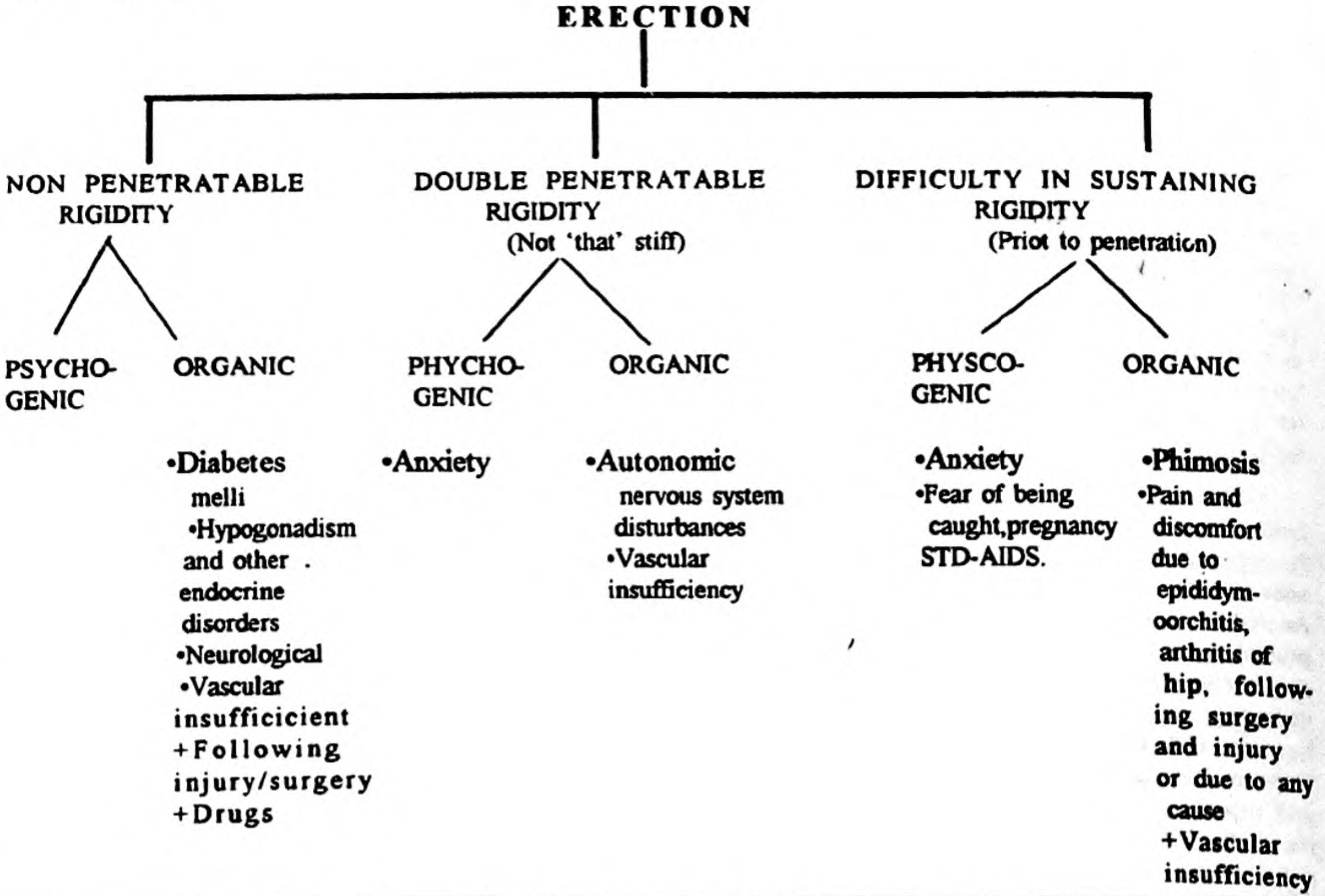
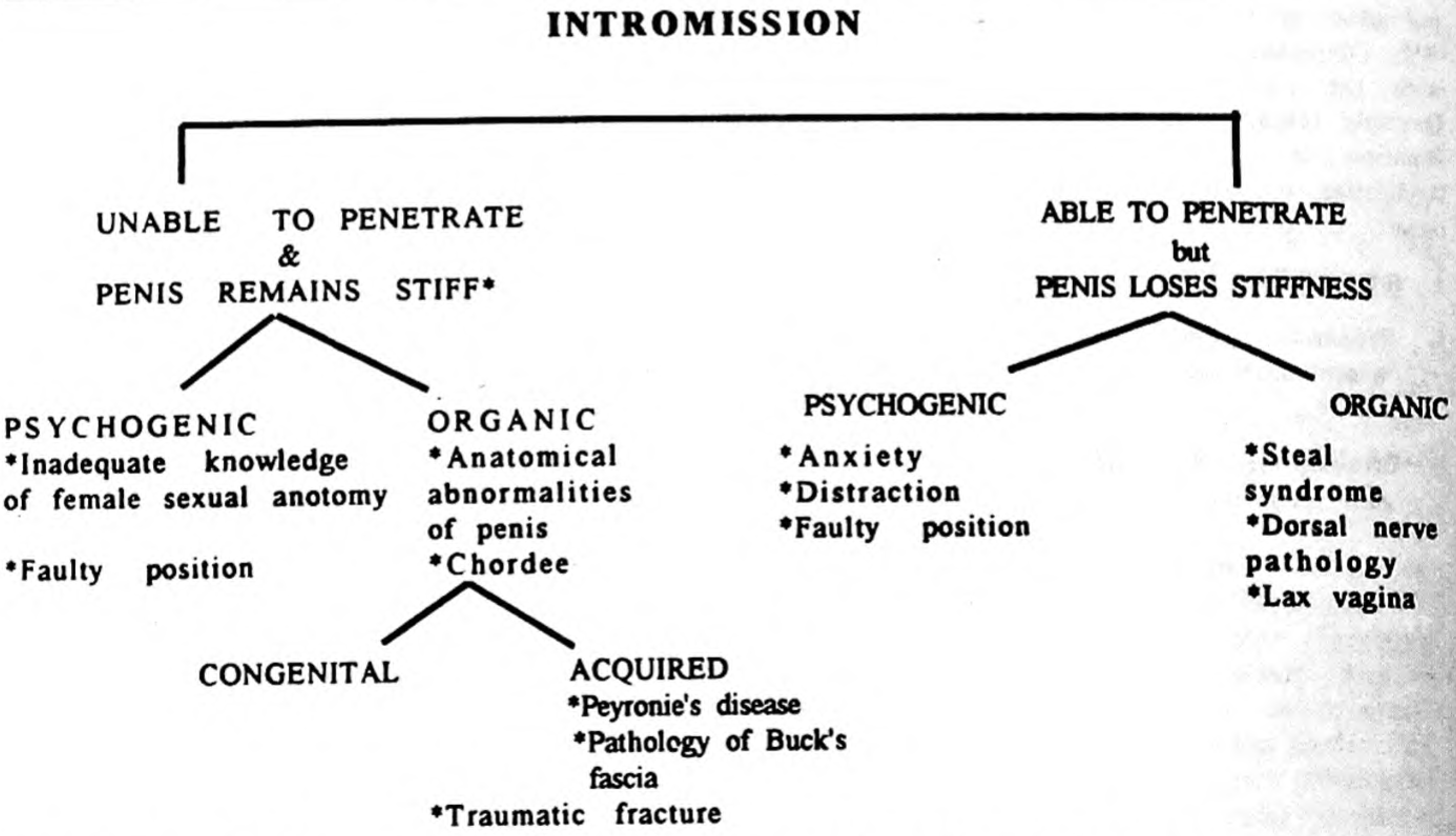
Flow chart.


ERECTION
NON PENETRATABLE RIGIDITY
PSYCHO-GENIC
ORGANIC
•Diabetes melli
•Hypogonadism and other endocrine disorders
•Neurological
•Vascular insufficicient
+Following injury/surgery
+Drugs
DOUBLE PENETRATABLE RIGIDITY (Not 'that' stiff)
PHYCHO-GENIC
•Anxiety
ORGANIC
•Autonomic nervous system disturbances
•Vascular insufficiency
DIFFICULTY IN SUSTAINING RIGIDITY (Priot to penetration)
PHYSCO-GENIC
•Anxiety
•Fear of being caught,pregnancy STD-AIDS.
ORGANIC
•Phimosis
•Pain and discomfort due to epididym-oorchitis, arthritis of hip, following surgery and injury or due to any cause
+Vascular insufficiency


INTROMISSION
UNABLE TO PENETRATE & PENIS REMAINS STIFF*
PSYCHOGENIC
*Inadequate knowledge of female sexual anotomy
*Faulty position
ORGANIC
*Anatomical abnormalities of penis
*Chordee
CONGENITAL
ACQUIRED
*Peyronie's disease
*Pathology of Buck's fascia
*Traumatic fracture
ABLE TO PENETRATE but PENIS LOSES STIFFNESS
PSYCHOGENIC
*Anxiety
*Distraction
*Faulty position
ORGANIC
*Steal syndrome
*Dorsal nerve pathology
*Lax vagina

*Vaginismus and obstructive vaginal pathology needs to be used out prior to planning the treatment

(Source: Indian journal of Clinical Practice)

"NUTRITION IN SURGICAL PATIENTS"

N.A. Wani (FRCS FICS)* W.S. Malik (M.S.)**

INTRODUCTION:

Recently there has been a great deal of interest in the nutritional status of patients. It is clear that there are numerous advantages in improving the nutritional state in a whole range of different medical and surgical conditions, by assisting, supplementing or replacing the patients normal diet. There is experimental evidence showing that malnourished patients are unduly susceptible to infections[1]. It has been shown that the length of hospital stay following major gastrointestinal operations was significantly reduced by early post operative feeding with elemental diet[2]. Overall, there seems to be sufficient reason to believe that more attention should be paid to the maintenance or improvement of nutritional status of malnourished patients.

NORMAL NUTRITIONAL REQUIREMENTS

An assessment of normal nutritional requirements in health is essential before we consider nutritional deficiencies and their corrections.

Normally, the human body requires a balanced intake of seven groups of nutrients viz: carbohydrates, fats, proteins, vitamins, electrolytes, water and trace elements. (Table-I). The immediate source of energy for the body is glucose. All three major nutrients—carbohydrates, fats, proteins, can be broken down to produce glucose. Of the 2500-3000 cals. required per day by an average healthy adult, nearby 50%/ is derived from carbohydrates. Glucose is readily available for intra cellular energy but little can be stored. Glucose, fructose and galactose are converted into glycogen which is stored in liver and muscles, available as a short term energy source. Glucose can also be derived from longer term energy reserves. Neutral fat is hydrolyzed into free fatty acids and glycerol and hence to glycogen and glucose. Although fat is a very good source of energy, a heavy dependence on it can be deleterious in certain situations due to an increase in Ketones and acids. Protein can also be metabolized and oxidized directly for energy but at the cost of Ketosis and muscle wasting.

Under normal healthy conditions, protein is utilized mainly for structurally purposes such as growth, replacement, repair maintenance of circulating proteins and enzyme manufacture. Excess amino acids are converted into lipids and since nitrogen is not stored in body, any surplus is excreted. An average healthy adult needs about 35g/day (0.5g/kg) of protein. The nitrogen content constitutes about 16% of ingested protein. In health, estimation of nitrogen intake and output provides a good index of protein intake and break down. About 90% of nitrogen is excreted in urine, and the rest eliminated Via faeces, hair and skin. Thus urinary urea concentration bears a close relationship to nitrogen excretion and may be readily measured to work out nitrogen balance.

The classic vitamin deficiency states are well known. Low serum levels of vitamins are often found in malnutrition without typical clinical changes and these deficiencies are frequently multiple.[3]

The importance of trace elements has been increasingly recognized in recent years, Specific deficiency syndromes are becoming known, especially when total parenteral nutrition is used without adequate trace element supplementation. Zinc deficiency produces skin rashes, hair loss and delayed wound healing[4]. Copper deficiency can produce neutropenia, megaloblastic anemia and bone demineralization[5]. Manganese, chromium, cobalt and selenium are among some other essential trace elements.

NUTRITIONAL ASSESSMENT:

An assessment of a patient nutritional status helps to provide an objective evidence of malnutrition and also helps in monitoring its progress, especially in severe sepsis or post operative states when nitrogen losses may vary from day to day.

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A simple scheme for assessing clinical malnutrition has been proposed[6] (Table-III).

This may also be used to monitor progress during hyperalimentation.

Triceps skinfold thickness, measured with calipers, gives an index of fat stores. If this figure is subtracted from midarm circumference then the result midarm muscle circumference, gives an index of muscle protein stores. Serum albumin levels are an index of visceral protein formation. Serum transferrin levels have also been used as an index of protein formation[7]. Urinary urea concentration bears a close and constant relationship with total urinary nitrogen and thus relates to the muscle breakdown occurring in the body[6].

In a healthy adult a neutral nitrogen balance exists (nitrogen in = nitrogen out). In catabolic states following surgery with sepsis a state of negative nitrogen balance (nitrogen in < nitrogen out) exists, indicating a faster tissue breakdown than can be replaced, there is a positive nitrogen balance (nitrogen in > nitrogen out), indicating tissue growth.

NUTRITIONAL REQUIREMENTS IN STARVATION AND DISEASE

Starvation:

During starvation the body utilizes some nutrients, that would normally be used for other purpose, for immediate energy. more monosaccharides are converted into glucose rather than glycogen or fatty acids. More fatty acids are oxidized in the liver. Amino acids are depleted of their nitrogen and converted for oxidation. Next, the muscle and liver glycogen undergoes glycogenolysis to glucose. After the first few days of starvation triglycerides are hydrolyzed and free fatty acids become the main energy source, leading to Ketogenesis.

As fat stores are depleted, in prolonged starvation, stored protein is used as energy source to maintain blood glucose. Body protein is converted into amino acids which undergo gluconeogenesis in liver, leading to muscle wasting. Although brain normally depends upon glucose for energy it can adapt to utilize ketone bodies. The state of enhanced ketogenesis seen in severe malnutrition can lead to ketoacidosis. In non-diabetics, an increased insulin secretion tends to inhibit this process and so prevents a dangerous build up of ketone bodies.

TRAUMA, BURNS, SEPSIS AND SURGERY:

These stress states can cause hypercatabolism, thus requiring more energy than ordinary or depleted nutritional supply can provide for the reactive processes to acute disease and for tissue repair. Fat and protein stores are broken down to provide energy for the stress state. The increased metabolic rate and nitrogen excretion rate and the higher blood glucose and insulin levels that occur in these stress states contrast with reduced energy expenditure, reduced nitrogen excretion, and low blood glucose and insulin levels that are present in starvation when conservation is the theme. Though fat mobilization is increased in both starvation and catabolic states, ketosis is only pronounced in starvation. The reason for this is the high insulin levels, that is found after surgery and trauma, which tends to inhibit lipolysis and the formation of ketone bodies. The insulin level is raised in response to high blood sugar levels, which in itself is secondary to the release of various stress related hormones, like catecholamines, corticosteroids, glucagon and growth hormone.

Whereas in starvation the energy requirements maybe met by 2000-3000 calories/day, in stress situations, that may be twice that figure. An average medical patient needs 8-12 g of nitrogen and 200 calories of energy source per day. In an uncomplicated post operative patient the nitrogen requirement may rise to 2-16g and that of calories to 2000-3500. Patients with severe hypercatabolic stress states over 16g of nitrogen and over 3500 calories/day may be required to maintain nutrition and nitrogen balance[6].

Normally 200 calories are required to utilize available protein in a 1 gm nitrogen source. In severe hypercatabolic states this ratio may rise to 500:1.

Disease states commonly associated with inadequate nutrient intake[8].

Post-operative states,
Adjunct to therapy in cancer patients.
Trauma and Burns, Sepsis, Renal failure, Hepatic failure.

G.I.T. Disorders:
Inflammatory bowel disease Short bowel syndrome
Pancreatic disease Adult Coeliac disease Biliary tract disease.

Table-I:

Average normal daily nutritional requirements

Calories	2000-300
Proteins	35g.

Electrolytes	(mmol)						Trace Elements			
Na	K	Cl	Po_4	Ca	Mg	Fe	Cu	Zn	Mn	Selanium
80	60	105	13	10-20	12-14	.18	0.3-0.4	0.2-.3	.017-.035	.0015

Table-II:

Vitamin Requirements:

					Recommended daily intake*							
Vit C	B1	Riboflavine	B6	Niacin	Pantothenic acid	Biotin	Vit A	Vit D	Vit E	Vit K	B12	Folic acid
(mg)	(mg)	(mg)	(mg)	(mg)	(mg)	(mg)	(IU)	(IU)	(mg)	(mg)	(ug)	(mg)
609	1.4	1.6	2	18	10	0.3	5000	400	30	700	5	0.4

* Food and Nutrition Board, U.S.A., for an adult male.

Table-III:

Evidence of Nutritional depletion[6]

Recent weight loss	>10%
Serum albumin	<35g/litre
Serum transferrin	<2g/litre
Triceps skinfold thickness	<10mm0 ;<13mm0
Midarm circumference	<23cm 0 ;<22cm 0
Lymphopenia	$1.2*10^9$/litre

Skin allergy e.g. negative Candida skin test.

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ORIGINAL ARTICLES:

EPIDEMIOLOGICAL CORRELATES IN PROTEIN ENERGY MALNUTRITION

G. M. MATTOO* G. N. SHAH**

SUMMARY:

612 children attending the Child Health Clinic of Community Medicine Department of Institute of Medical Sciences Srinagar in the year 1991 were evaluated for malnutrition; the role of various epidemiological correlates was studied in relation to malnutrition and impact ratio of each factor was calculated. Overall prevalence of malnutrition was found as 46.57%. Birth order, maternal addiction, maternal illiteracy bore direct relation whereas birth interval and per-capita ·income per month had indirect relationship with malnutrition. Moreover malnutrition was found more in females, rural cohort and children in nuclear families.

INTRODUCTION

Malnutrition among pre-school children is a complex phenomenon being influenced by multiple factors in multiple dimensions. It forms a matter of great concern especially in developing countries like India, where its prevalence of moderate to severe forms has been estimated as 45% in the age group of less than 5 years from NNMB data. (1). Keeping in view the magnitude of the problem, need is left to identify the role of different factors, be they nutritional or non-nutritional, which bear significant impact on such morbid conditions which if controlled or eliminated could reduce the magnitude of this problem to a considerable extent.

In Jammu & Kashmir since need based studies to evaluate growth and nutritional status of pre-school children are limited in number, an attempt is made to assess the manigtude of malnutrition in this age group and the role of which certain epidemiological factors play in its causation.

MATERIAL AND METHODS

The study covered 612 children who attended the Child Health Clinic of the Department of Community Medicine, SKIMS during January 1991 to January 1992. The information regarding epidemiological correlates was acquired from mother or one accompanying the child through a pretested oral questionnaire on cross-sectional basis. For identification of malnutrition the anthropometric measurements were taken and finally classification was made following Harvard Standard (weight in relation to age of the child) modified by Indian Academy of Pediatrics. (4) The weight of the child was taken on a platform beam balance.

The data finally was subjected to statistical procedures to get inferences and impact ratio in respect of each attribute was calculated.(3)

OBSERVATION & DISCUSSION:

The present study showed an overall prevalence of 46.75% malnutrition comprising of 23.20% grade I, 12.75% Grade II, 7.35% Grade III and 3.27% grade IV. The study also depicted a significant positive association ($P< 0.025$) of malnutrition with the age of the child being minimum 42.61% in infancy and maximum 53.28% in the 4-5 years age of group (Table-1).

This overall figure is much below the value depicted in other studies. Pralhad Rao et. al (2) reported a prevalence of 80% malnutrition in poor communities of India, whereas Dwivedi et. al (6) reported a prevalence of 63.4% in urban slum children of Madya Pradesh. The children in the present study predominantly were from urban areas and from middle class families could account the lesser percentage of malnutrition present in the earlier years of life.

The socio obstetric correlates studied were birth order, Sex of the child, birth interval (in months) and maternal addiction (Table-2).

Birth order showed a significant association ($P<0.0001$) with malnutrition being minimum (33.02%) among the first order children and maximum 70% among those with birth order of above 3. The impact ratio was 0.48 for birth order 1-3 and 0.43 for birth order more than 3. These findings are almost similar to those found by Dwivedi et. al (192). (6)

Female children also were found to have

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TABLE-I PREVELENCE OF PEM IN RELATION TO AGE (Yrs) OF CHILD.

S NO	Age	Total children studied No.%	PEM G-I No.%	G-II No %	G-III No.%	G-IV No %	TOTAL No.%
1	<1	230 37 67	59 25.65	20 8.70	13 5.65	6 2.61	98 42 61
2.	1-2	102 16.76	19 18.63	8 7.84	9 8.82	6 5.88	98 41.18
3.	2-3	78 12.75	16 20.51	10 12.82	7 8.97	5 6.41	38 48.72
4.	3-4	80 13.07	18 22.50	12 15.00	9 11.25	3 3.75	42 53.28
5.	4-5	122 19.93	30 24.59	28 22.95	7 5.74	- -	65 53.28
TOTAL:		612 100.00	142 23.20	78 12.75	45 7.35	20 3.27	185 46.57

Remarks: $X^2 = 6.13$

TABLE-II: IMPACT OF SOCIO OBESTETRIC CORRELATES ON PEM.

S.NO	Correlate	Attribute/ Variable	Total Children studies (No.%)	PEM No.%	Impact ratio	ST. imference
1.	Birth order	1	212 34.64	70 33.02	-	
		1-3	310 50.65	152 49.03	0.48	X 2
		>3	90 14.71	63	0.43 p<.001	36.27 Sig.
2.	Sex of Child	Males	289 48.69	87 29.19	x 2 -d	
		Females	314 51.31	198 63.06	1.16	1=70.45 P<.0001 sig
3.	Birth interval months	<18	68 11.11	48 70.59	0.23	x 2 d
		18-24	322 52.61	185 47.45	1.45	p<.0001 sig.
		>24	222 36.28	52 23.42	-	
4.	Maternal	Smoking	43 6.86	29 69.91	0.54	x 2 df1=9.16
		Non-Smoking	570 93.14	256 44.91	-	p<.0001 sig.

x Impact ratio calculated between two consective stages.

TABEL-III IMPACT OF SOCIO ECONOMIC CORRELATES ON PEM

S.NO Correlate	Attribute/ Variable	Total children No. (%)	Pem No.(%)	Impact ratio	St. inference
1.Residence	Rural	208 33.99	145 69.71	1.01	x^2 d 1=67.83 P<.0001
	Urban	404 66.01	140 34.65	-	
2.Type of family	Nuclear	188 30.72	117 62.23	0.57	x^2 d f1=26.76 P<.0001
	Joint	424 69.28	168 39.62	-	
3.Per capita income (Rs)	<100	164 26.8	102 62.20	0.24	x^2 d f2=41.04 P<.0001)
	100-250	250 40.85	125 50.00	-	
	>250	198 32.25	107 29.29	-	
4.Maternal litracy	Literate	358 58.50	107 29.29	-	x^2 d f1=96.47 P<.0001
	Illiterate	254 51.50	178 70.08	1.34	

higher prevalence of malnutrition than their male counterparts, the difference being statistically significant (p<0.0001) and impact ratio was 1.16. The findings were in resonance with the findings of Maya Chowdhary and Vishweshwara Rao (1983) in their study in Jaipur Rajasthan and Dwivedi et al (1992) in their study in Madhya Pradesh.(5,6)

Birth interval (between the evaluated child and its antecedent) showed an inverse highly significant (P<0.001) relation, with the prevalence of malnutrition being maximum 70.59% among children with birth interval of 18 months or less and minimum 23.42% among those with the interval of 24 months or more. The impact ratio with the 18 months or less birth interval being 0.23% and with the birth interval of 18-24 months being 1.43. Similar findings have also been reported by Dwivedi et al (1992) and Ahmad.P et al (1983) in their respective studies. (6,7)

Prevalence of malnutrition was also found to be significantly higher (P<0.0001) in children whose mothers were smokers (44.91%) (the other addictions being nil among females in this part of the country) with an impact ratio of 0.54. But since the number of smoking mothers was very small (6.86%) nothing concrete could be drawn from this observation.

In the present study 34% of children were rural and 66% were from urban areas, Malnutrition was found significantly more prevalent (P<0.001) in rural children (69.71%) than in urban children (34.65%) with an impact ratio of 1.01. Similar findings have been reported by Maya Chowdhary and Vishweshwara Roa from their study in 1982.(5).

Type of family when studied showed that malnutrition was more prevalent in children from Nuclear families (62.23%) than in those from joint families (39.63%) with a significant statistical difference (P<0.0001). The impact ratio calculated being 0.51. The difference could be explained on the fact that in nuclear families, the mother remains busy with her household, neglecting the child, whereas in joint families all household tasks including the rearing of children are shared by other members of the family.

Per capita income was also seen to have a significant association with malnutrition ($P<0.0001$) with 62.2% children from the lower income group suffering from malnutrition as compared to 50% of children from the slightly higher income group. Dwivedi et al & Maya Chowdhary and Vishweshwara Rao also reported similar association in their studies. (6,5).

Maternal literacy also showed a significant relation ($P<0.0001$) with the prevalence of malnutrition with only (29.89%) of children from literate mothers compared to 70.80% of children from illiterate mothers suffering from malnutrition. Similar results have been reported from other studies. (4,5).

CONCLUSION:

From the present study it can be concluded that malnutrition is quite prevalent in this part of the country and forms an important problem to be dealt with.

The magnitude could be controlled by means of interventions, in socio-economic and socio-obstetric characteristics through primary health care approach.

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Information to author continued

Abstract and key words

The second page should carry an abstract of not more than 150 words stating the purpose of the study or investigation, basic procedures, main findings (state specific data and their statistical significance, if possible), and the conclusions in principal.

Text

The text of observational and experimental articles is usually-but not necessarily-divided in to section with the headings, introduction. Methods, Results and Discussion. To clarify their content, especially the results and discussions sections long articles may need subheadings with some sections.

Acknowledgments

The persons who have made substantive contributions to the study can only be acknowledge. It is author's responsibility to obtain written premising from the persons whose name have been acknowledged because readers will infer their endorsement of the data and conclusion.

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References should be numbered consecutively in the order in which they are first mentioned in the text. Identify references by arabic numerals in text, tables and legends (in parentheses). Reference cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text, of the particular table or illustration. Include among the reference, manuscripts accepted but not yet published, designate the journal followed by in press in parenthesis. Information from manuscripts submitted but not yet accepted should be cited the text as unpublished observations in parenthesis.

Continued on page 28

PREVALENCE OF GOITER IN SCHOOL CHILDREN IN KASHMIR VALLEY

A.H Zargar, (D.M)*, J.A. Shah, (M.D), M. Muzaffar Mir, (Ph.D), B.A. Laway, (M.D), S.R. Masoodi, (M.D), N.A.Shah (M.D)

INTRODUCTION

Iodine deficiency is an important public health problem of world-wide distribution, resulting in endemic cretinism, characterized most commonly by mental deficiency, deafmutisim, spastic diplegia and lesser degrees of other neurological defects. The presence of goiter is a sensitive indicator of iodine deficiency (1,2). It is vital that the magnitude of goiter prevalence be quantified and its relationship with iodine deficiency be determined so that strategies could be developed to eradicate this massive public health problem. This study was conducted with a view to determine the prevalence of goiter in school children in the Kashmir Valley in the Indian sub-continent and quantitate the iodine status by its urinary estimation in the surveyed population.

POPULATION AND METHODS

This study was conducted on school children of Kashmir Valley in the age group of 5-15 years. Students of both the sexes were included. The study was conducted by adopting multi-stage sampling procedure. A total of 10,196 subjects were examined for the presence of goiter. The presence and magnitude of thyroid enlargement was studied as recommended by Karmarker et al (4). Statistical analysis was done by Chi-square test.

RESULTS AND DISCUSSION

The study was conducted in 210 randomized villages of Kashmir Valley studying a total student population of 10,196 in the age group of 5-15, which included 5,676 boys and 4,520 girls. Out of total 10,196 students studied, 4,609 (45.2%) had different grades of thyroid enlargement (Tabel-1). There were only two cases of grade-III goiter and none had grade IV thyroid enlargement.

The overall goiter prevalence increased with age with prevalence of 30.26% in the age of group 5-12 years and 50,67% in the age of group of more than 12 years (Tabel-2). In the 202 urine samples, iodine excretion ranged from 6.25 to 103.6 micrograms per gram of creatinine with mean ± SEM of 49.60 ± 3.55 micrograms per gram of creatinine excreted iodine deficiency is the principal etiological factor in endemic goiter, and uninary iodine exsertion per gram of creatinine in the single urine sample has been successful used in the estimation of iodine deficiency or sufficiency states (5).

Iodine deficiency related goiter continues to be a significant public health problem throughout world including Europe (6). Studies carried in various parts of India have related prevalence of goiter with iodine deficiency states (7). There are innumerable pockets in the goiter belt of Himalayan and sub-Himalayan regions, where the prevalence of goiter among school children varies from 55 to nearly 100 percent (8). The urinary iodine excretion in our study was 49.602±3.55 microgram per gram of creatinine which suggest that Kashmir valley is an area of moderate iodine deficiency and measures need to be taken to rectify this public health problem of major importance.

Tabel-1

Prevalence of different grades of goiter

Total Population Studied 10,196

S.No (%)	Grade of goiter	No. Of children with goiter	Prevalence
01	OB	1640	16.08
02	I	2208	21.66
03	II	759	7.44
04	III	02	0.02
Total		4609	45.20

---

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Table-2
Overall goiter prevalence according to age

Age group (years)	Total population studied	OB No./%	Grade of Gotier I No./%	II No.%	Total No. No./%	Statistical inference
<6	1041	142 (13.64)	139 (13.35)	34 (3.27)	315 (30.26)	$X^2df_4=266.44$ P <.001 Highly significant
6-8	1851	254 (13.72)	273 (14.75)	98 (5.29)	625 (33.76)	
8-10	3007	563 (18.72)	719 (23.91)	215 (7.15)	1497 (49.78)	
10-12	2189	344 (15.71)	553 (25.35)	205 (9.37)	1102 (50.34)	
>12	2108	337 (15.99)	524 (24.86)	207 (9.82)	1068 (50.67)	
Total	10196	1640 (16.08)	2208 (21.68)	759 (7.44)	4607* (45.20)	

Two cases of the great III Goiter are not indicated in the Table.They were observed in >12 Yr. age group.

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ORIGINAL ARTICLES:

HYPOFRACTIONATION V/S DAILY FRACTIONATION RADIATION TREATMENT FOR UNRESECTABLE NON-SMALL CELL LUNG CANCER.

NAQASH AA; SANYAL B; KHARADI MY; GURU AA*

The overall incidence of lung cancer has been on the rise and look so even in this part of the country, it seems to run parallel to oesophageal cancer. Since the overall results have been dissimil with either modality, this study was undertaken with an aim to improve local regional control in non-small cell lung cancer.

Sixty patients with advanced locoregional non-small cell carcinoma of lung were treated with two different fractionation schedules of radiation therapy. The patients were divided into two groups. Thirty patients were treated with conventional fractionation schedule of daily fraction of 200cGy to a total tumour dose of 6000 cGy/30 fractions/6week (NSD = 2192 ret). This comprised of control group and study group constituted equal number of patients, were treated with 680 cGy once a week to a total tumour dose of 4080cGy in 6 weeks (NSD = 1800 ret). Patients in both groups were comparable in age, sex, smoking habits, histology, grade of the tumour and the stage of cancers.

Complete response (total disappearance of the total tumour) at 3, 6, 9 months was higher in study group (23.3%, 13.3% and 10% respectively) compared to the control group (13.3%, 10% and 6.7% respectively); however these differences were statistically insignificant ($p>0.5$). There was no significant difference ($p>0.5$) in overall survival rates at one year between study and control groups (43.3% Vs. 36.7% respectively.

There was no major toxicity and the patients treatment tolerance was acceptable. Radiation therapy is very effective in local tumour ablation and since both fractionation schedules are comparable in effectiveness, once a week therapy emerges as a convenient and practical modality of treatment in inoperable non small cell lung cancer.

(DEPARTMENT OF RADIATION ONCOLOGY AND CARDIOVASCULAR &THORACIC SURGERY

S.K.I.M.S Soura, (SRINAGAR KASHMIR)

INTRODUCTION:

Lung cancer with its increasing incidence and high mortality rates continues to be a major health problem throughout the world despite the tremendous advances with the oncological sciences have achieved. Surgery is the treatment of choice in early stage lung cancer. Unfortunately less than one fourth of patients with non-small cell lung cancer have disease considered rescuable at the time of diagnosis. Even when disease in considered to be surgically operable, the 5 year survival of patients undergoing tumour resection is only about 25% and 5 year survival of all patients is less than 10%. This figure has not changed substantially in the paste decade. Early stage constitutes only a fraction of patients because of late presentation and overt diagnosis, majority of patients present in advance stage and are inoperable (3). In patient with locally advanced non-small cell carcinoma of lung, radiation therapy is effective in achieving local tumour control (6). However, the optimum time, dose, fractionation schedule for treatment is yet to be involved. The basic of conventional fractionation in radiotherapy is merely empirical. During past few decades, the conventional fractionation regimen using five fractions per week for 5-7 weeks has been established for the most of malignant disease (4). In past two decades, the resurgence of interest in hypofractionation has arisen and has been found to be as effective as conventional treatment regimen (I).

This study analyses 60 cases of locally advanced non-small cell lung cancer treated by two different schedules of fractionation regimes of radiation therapy to compare efficacy and tolerance of treatment and over all results.

MATERIAL AND METHODS:

A total of 60 patients with histopathologically proven cancer of non-small cell variety were taken up for this study between July 1988 to December 1989. All patients were subjected to initial evaluation with hematological, biochemical, radiological and radionuclide investigations. The patients were treated with conventional fractionation radiation treatment which constituted control group whereas other thirty patients were treated with hypofractionation treatment regimen constituting study group. The age distribution of the patients depicts the mean age of patients in control group 54±7.3 years whereas in study group it was 52.6±6.8 years (table-I).

TABLE-I:

DISTRIBUTION OF THE CASES ACCORDING TO AGE

AGE GROUP (IN YEARS)	STUDY GROUP No.of pats.	STUDY GROUP %age	CONTROL GROUP No.of pts.	CONTROL GROUP %age
0 - 40	02	06.67	02	6.67
40 - 45	01	03.33	02	6.67
45 - 50	08	26.67	04	13.33
50 - 55	06	20.00	05	16.67
55 - 60	09	30.00	11	36.66
60 & above	04	13.33	06	20.00
Mean Age ±sd	(52.67 ± 6.86)		(54 ± 7.33)	

t = 0.74, $p > .05$
(Not significant)

(Table-II).

STAGE AND HISTOLOGICAL DISTRIBUTION

STAGE NO.	STUDY GROUP NO.	STUDY GROUP %AGE	CONTROL GROUP NO.	CONTROL GROUP %AGE
I	0	-	0	-
II	09	30	11	36.67
III	21	70	19	63.33
HISTOLOGY				
i) Squamous cell carcinoma	28	93.33	30	100.00
a) Well differentiated squamous cell carcinoma	26	86.66	26	86.66
b) Moderately differentiated squamous cell carcinoma	-	-	-	-
c) Poorly differentiated squamous cell carcinoma	02	6.67	4	13.34
ii) ADENOCARCINOMA	02	6.67		

The majority of the patients were overstayed - stage III, 70% in study group and 63.3% in the control group. Squamous cell carcinoma with high grade constituted the bulk in the both groups (Table II)

Patients in control in group were treated with megavoltage 60 external beam radiotherapy CO with conventional fractionation. The dose was 200cGy/day, 5 days a week to a total tumour dose of 60 Gys by two parallel opposing A_P and P_A portals. Spinal cord was shielded at the tumour dose of 40 Gys. Patients in study group were treated with radiation therapy with the single fraction of 680 cGy delivered each week with two parallel opposing A-P and P-A fields for 6 weeks to a total tumour dose of 4080 cGy. Shielding of spinal cord was done after 4 fractions at the dose of 2720 cGy. Thais was a precautionary measure to avoid late spinal cord damage. This dose was well within the tolerance limits of spinal cord. All the patients were assessed at weekly intervals for tumour response, mucosal reactions, skin reactions and systemic effects during the treatment and then at monthly for first six months and every three months next.

RESULTS:

The treatment results were evaluated with respect to response both clinically as well as with radiological regression of primary tumour and over all survival. The radio responsiveness of the primary tumour was measured by percentage of regression of pulmonary shadow on chest skiagram and at least two dimensional measurements were obtained at the end of one month after treatment and thereafter every three months. The response was classified into three categories-complete response (CR) - total disappearance of tumour, practical response (PR) -decrease is more than 50% in the product of at least two diameters and the response is to be sustained at least for more than two months whereas term no response (NR) - situations like, tumour decreased by less than 50% or there was a progression of the disease.

The overall response was 70% (26.67% CR and 43.33% PR) in the study group as compared to 56.67% (23.33% CR and 33.34% PR) in the control group. At the end of one year, the results in two groups were looked at the respect to local control and survival and there was statistically no significant of difference between the two groups as for as local control is concerned.

The median survival for the patients in study groups was 11.5 months and 9.7 months for control group, whereas one year survival was 43.3% and 36.6% respectively. This difference was also insignificant.

These patients were monitored closely for acute and late toxicity. Once a week radiation fractionation regimen was found to be well tolerate and it was completely devoid of acute reactions like oesophagitis which was noted in the patients treated with conventional radiation treatment. the patients were observed for late reactions like fibrosis, cardiomyopathy and myelopathy. None developed myelopathy in either group. Four patients in study group (7.5%) and two patients in control (6%) developed cardiomyopathy. However, it is difficult to implicate radiation alone as causative agent in these patients. Lung fibrosis at 12 months was 33% in control group and 50% in study group.

DISCUSSION

Non small cell lung cancer is a major health problem with 90% mortality and median survival of less than a year. The majority of the patients are unresectable and radiotherapy is being widely used to control local tumour. However, optimal time, dose and fractionation schedule is yet to be evolved. Cox et al, 1972 (2) reported 30-50 percent local tumour control with conventional fractionated external beam radiotherapy in unresectable lung cancer, whereas hypofractionation has been used in many centres to reduce the patient load on the machine and for the convenience of the patients and staff (8). The Berlin experience indicated that this approach achieved acceptable survival, toxicity and response which were as good as with conventional fractionation schedules (7). Weekly fractionation schedule have its own radiobiological advantages as one week's interval between the fraction would provide better opportunity to normal tissues for repair and enough time for removal of cell debris resulting in tumour regression, revascularization and subsequent improved radioresponsiveness because of better oxygenation (5).

In the present study, a comparison was attempted between patients treated with 60Gys of continuous radiation with conventional fractionation schedule and the patients treated with once a week irradiation of 60cGy to total tumour dose of 4080cGy in 6 weeks. In terms of local tumour control, survival and tolerance, the local response seen with once a week schedule has been found encouraging and our findings are in agreement with those of Slawson et al, 1988 (9) who reported complete response rate of 86% (CR 26% + PR 60%). However, on analyzing our result statistically, there was no significant difference between two regimes in terms of local response.

All these patients were followed for a period of one year and those who were lost to follow up were presumed to be dead keeping in view the nature of illness and advance disease status. The survival pattern was comparable in both the groups without any significant difference at any stage. There were practically no acute reactions in the study group.

Nevertheless hypofractionation schedule is believed to lead to more severe late complications. In the present study, the patients were followed up for a period of one year and during this period, there was no significant difference even in terms of late complications like lung fibrosis and no case of radiation myelitis was noticed in either group.

However, long term follow-up is warranted to reach a conclusion that hypofractionation is as safe as conventional fractionation schedules in terms of late complications.

It is a concluded from this study that once a week radiation is as effective as conventional fractionated radiotherapy in unresectable non-small cell lung cancer and is well tolerated regimen. In addition, it has tremendous advantages as it suits the patient's convenience and reduce the load on the teletherapy machines.

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Type each table on a separate sheet using double space. Supply a brief title for each table numbering consecutively. Give each column short of abbreviated headings. All nonstandard abbreviations that are used in each table should be explained in footnotes. For footnotes, use the following symbols in this sequence : * , +, +, \$, II, 88< ++, ... Identify statistical measures of variations such as SD and SEM. Omit internal horizontal and vertical rules. Cite each table in the text in consecutive order. Obtain permission and acknowledge fully, if you use data from another published or unpublished source.

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Sd/-Dr. G. M. Malik

BRIEF REPORTS:

INTRACIVATARY PULMONRY MASS IN A PATIENT WITH AIDS

Khurshid A.Guru*, Imran Mirza**, Leonardo Barret***, F.A KHAN****

A 34-years-old male intravenous drug abuser presented with cough and shortness of breath of two weeks duration. The cough was associated with blood streaked sputum. Patient had documented AIDS of seven years duration, with a CD_4 count of 59(normal 390-1460/cumm). Six months prior to the present admission,he had presented with a pulmonary infection which was felt to be afungal in nature based on sputum culture which grew Aspergollus fukmigatus. His brief trial on amphotericin was discontinued after 20 days because of adverse effects. he was maintained on fluconazole, which he continued to take. His physical examination revealed no respiratory distres. He had pallor, clubbing of fingers with associated scanty left-upper lobe crepitations on auscultation. All other systems were unremarkable. Chest x-ray findings revealed a 3 x 2 cm cavity in left-upper lobe with smooth margins and an intracavitary mass (Fig.1). EKG results were normal. Arterial blood gas on room temperature revealed a pH of 7.4; $PO_2$98 mmHg; $PCO_2$30 mmHg; $HCO_3$32. His WBC was 11.1 k/mm^3 with 50 % neutrophils, 17% lymphocytes. Routine sputum cultures did not grow any bacteria or fungi. He was sbsequently bronchoscoped and no evidence of pneumocystis carinii was found. In view of the lack of definitive diagnosis, patient was reffered to Thoracic Surgery.

DIAGNOSIS: Intracavitary Aspergilloma.

Patient had a left-upper lobectomy, along with a left-lower segmentectomy.
The resected gross specimen measured 10 x 18 x 5 cm. The pleural surface showed a moderate degree of anthracotic mottling near the opening of the superior lobe. There was a rounded cystic structure measuring 6x4x4cm. On opening, it revealed a thickwalled cavity with corrugated margins filled with a tancoloured cheesy material.

Histopathlogic examination revealed a fibrous cyst wall filled with fungal elements and necrotic debris. The fungal elements composed of branching septate hyphae and spores characteristic of aspergillosis (Fig.2). In addition, there were areas of acetic and organizing pneumonia and giant-cell infiltration. Representative section from the remaining lung also showed fungal invasion of the terminal bronchioles and the pneumonic lung parenchyma.

Eight years after the intial diagnosis of AIDS, and 14 months after resection of the left-upper lobe aspergilloma, patient is alive and attends the outpatient clinic on a regular basis.

Aspergillus species are ubiquitous in the environment and only rarely cause significant disease in healthy hosts. The common risk factors for opportunistic infections are corticosteroids, broad spectrum antibiotics and other possible risk factors include diabetes, alcohol abuse,chronic lung disease, cavitary lung disease, viral infection, etc. Relationship between HIV infection and aspergilllosis is evolving as patients live longer and develop granulocyte dysfunction, either due to the HIV infection or the concomitant usage of steroids or AIDS-related therapies like Zidovidine and Ganciclovir which can result in neutropenia. The overall incidence of Aspergillus in HIV patients ranges from 0.16%-5 out of 3,170 patients with AIDS reported to CDCbetween MAY 1983-JUNE 1984, to 0.9% from out of 470 patients at the University of Pennsylvania from july 1988-December 1991 (2). When Aspergillus develope in AIDS patients, it can range from invasive to the localized form of Apergillus. The lung has been dicumented as the most common site for Aspergillus in AIDS patients, with hemoptysis as the most common and life-threatening symptom occuring in 60-80% of cases. A few may also present with productive cough, weight loss and finger clubbing (1). Unusual variants have presented with extensive necrotic brochitis which somtimes leads to fatal hypoxia. Other features described include maxillary sinusitis, skin infections and

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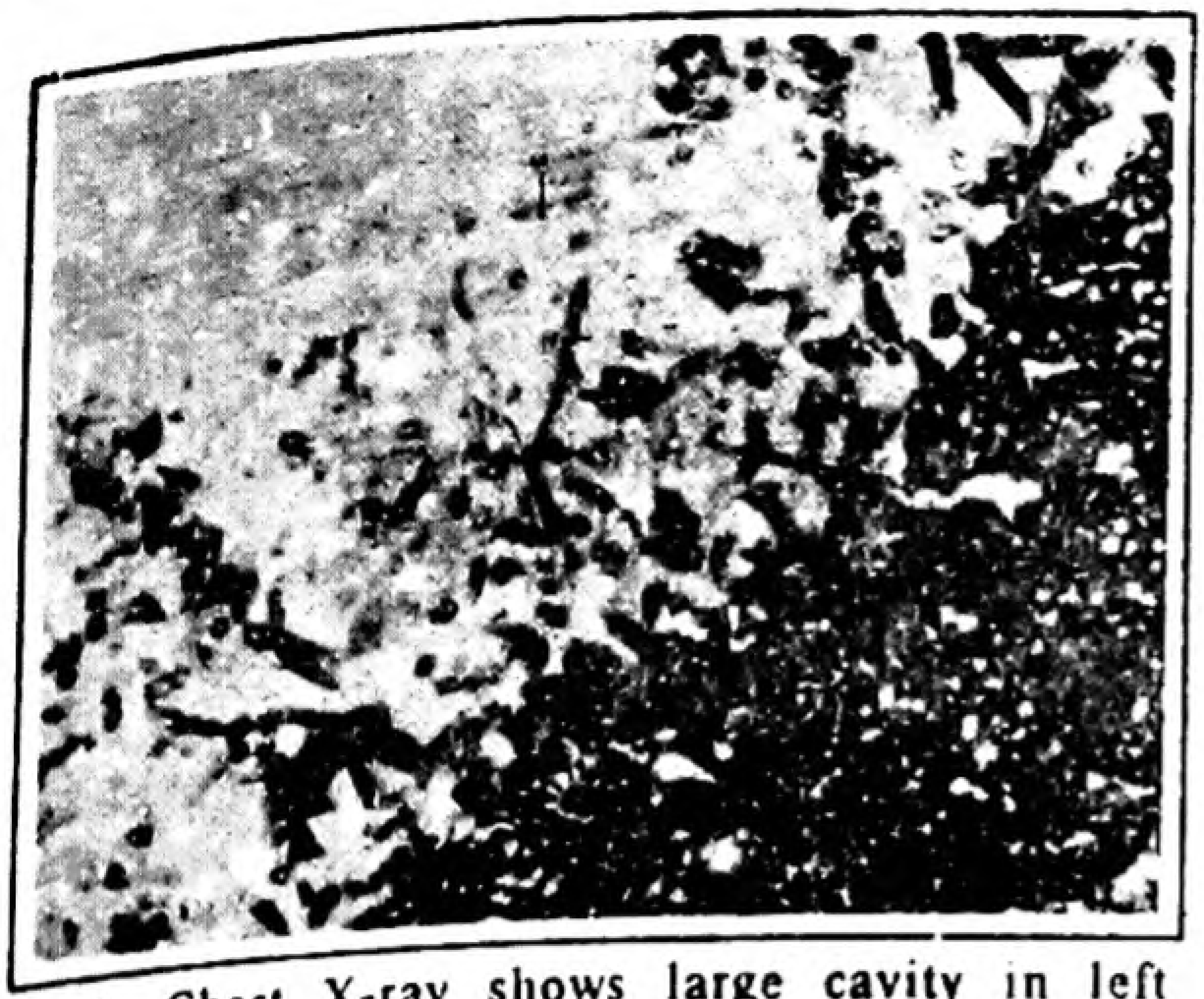

Fig.1. Chest X-ray shows large cavity in left upper lobe with an intracavitary mass.

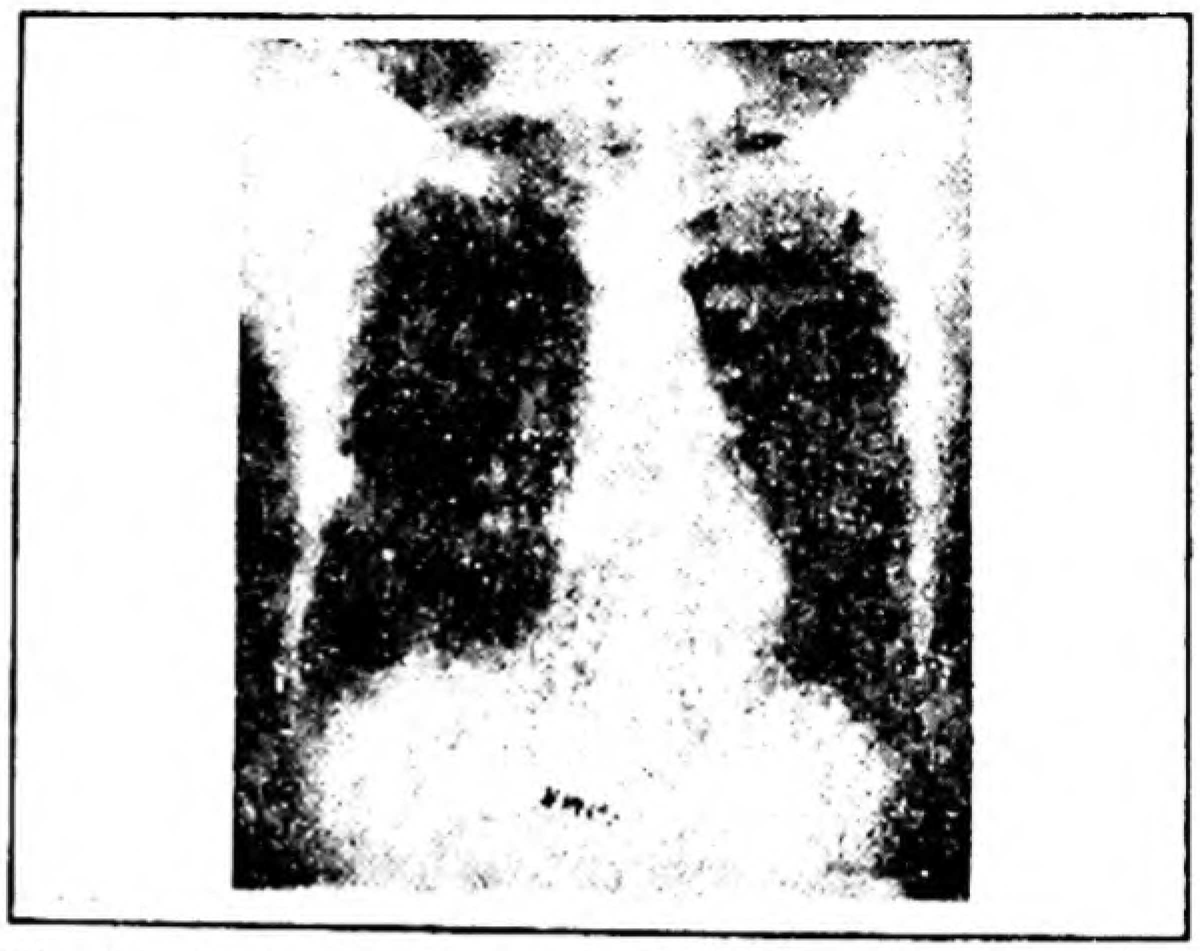

Fig.2: Spores and hyphea characteristic of Aspergilus within the cavity.

very rarely endocarditis, esophagitis, brain abscess and renal cysts (4).

Pulmonary aspergillosis presenting as cavity lung disease, particularly in upper lobe, was present in 13 of the 36 patients with Aspergilluso reported (2). The presence of an intracavitary mass was noted in only three out of these patients (2). Aspergilloma forms when the fungus grows on the walls of a lung cavity and the mycetoma with the debris becomes detached to form a concretion of amorphous material containing tangled septate hyphae with altered blood elements (1).The radiographic feature include an intracavity.solid, rounded mass sometimes associated with positive-sputum culture for Aspergillus fumigatus. Blood cultures are rarely positive for Aspergillus growth (3). The major complication of this aspergilloma massive recurrent hemoptysis,and frequent mild hemoptyic episodes may also occur. Of the 13 patients with cavity lung disease caused by Aspergillus reported by Miller, six had hemoptysis which was fatal in five. This fact should be borne in mind when decision regarding management are made in HIV patients with cavity lung disease related to Aspergillus. The pathogenesis of hemoptysis involves frictional forces within cavity wall, hemolytic endotoxins or proteolytic enzymes from granulocytes (5).

In summary, we have belive this case report highlights the following clinical features.

a. Aspergillus as a primary opportunistic is uncommon in HIV-infected patients.

b. As patients with HIV disease live longer, receive treatment alters granulocyte function, they become more susceptible to Aspergillus infection.

c. In the Hiv patients, Aspergillus may range from a cavitary lung disease, with occasional intracavitary mass, to focal alveolar opacity, bilateral opacity and invasive aspergillosis.

d. Presence of cavitary from Aspergillus in HIV patients carries a serious prognosis, and early surgical therapy may be beneficial in preventing the often lethal complication of hemoptysis.

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GUNSHOT WOUNDS OF THE PREGNANT UTERUS:

"BABY WINS THE BATTLE"

Mufti M.A*, Wani M.A*, Afshan A.**, G.H Bhat***, Mumtaz Din***, Mir N.A****, A.H Naqashbandi*****

Introduction:

Penetrating abdominal wounds during pregnancy may result from a variety of objects animal horns, files, pitch forks, scythes, sickles and knives(1). While these are of historical interest, the incidence of gunshot wounds to the pregnant uterus is increasing as a result of the increasing use of handguns and crime,, increased urban strife and the exposure of civilian population to Guerilla warfare. The real incidence of gunshot wounds during pregnancy probably far exceeds to the reported rate. A total of 88 cases of gunshot wounds to the Pregnant uterus have been reported in 1980[4].

Here are **reported two** cases of gunshot wounds of **the pregnant** uterus and their management.

Case Report - 1:

A 30 years old primi-gravida at 28 weeks gestation was received in the emergency room of S.M.H.S. Hospital, Srinagar about one hour after sustaining a bullet wound of the upper abdomen approximately 5 cms, above 3 cm to the left of the umbilicus (in the epigastric region). There was moderate bleeding from the wound of entry, no wound of exit.

Physical examination revealed a healthy women in moderate distress with unstable vital signs - pulse 130/min. regular, respiration 26/min, Bp:100/70 mmHg and temperature 37°C. The abdomen was soft and moderately tender with a bullet felt subcutaneously in Right iliac fossa (RIF). Bowel sounds were sluggish. The uterus was soft, symmetrically enlarged with the fundus about 28 cms above the symphysis pubis. Fetal heart sounds were absent. Per-Vaginum examination revealed a closed and unaffected cervix with no blood or amniotic fluid present in the vagina. The presentation of the fetus was cephalic.

A foley's catheter returned clear urine from the bladder. Laboratory data were : hematocrit 30%. No roentgenograms of the abdomen were taken.

The patient was prepared for surgery. Under endotracheal general anesthesia, a celiotomy was carried out through a midline infraumbilical incision. Approximately 800ml of blood were present in the general peritoneal cavity and the pelvis. The operative findings were :

1. 1cm entry wound on the posterior aspect of the uterus near its fundus with an exit wound in the right lower lateral aspect of the uterus. Both wounds had minimal bleeding from their edges.

ii. An exit wound in the parities (abdominal wall muscles) and the blood lodged subcutaneously in the RIF.

iii. Multiple perforations (three in number) were found in the small gut (distal Jejunum) from where minimal amount of feculent matter had soiled the peritoneal cavity.

A lower segment transverse caesarian section was performed with the delivery of a stillborn male body weighing 8 lbs. the baby had a 2cm. long superficial lacration of the right ear. The caesarian section wounds was closed in two layers. The uterine wounds were closed after proper debridement. The small gut bearing multiple perforations was dealt with by resection and end-to-end anastomosis in 2 layers. The peritoneal cavity was lavaged with N-saline, mopped dry and a corrugated rubber drain (CRD) placed near the anastomotic site. After closure of the abdomen in layers, the bullet lodged subcutaneously in RIF was removed by a small incision over the bullet. The patient received 900ml of whole blood and was put on prophy-

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lactic broad spectrum antibiotics comprising I/V Cefotaxime and I/V Metronidazole. The recovery of the patient was uneventful and she was subsequently discharged from the hospital.

Case Report - II :-

A 35 years old primigravida at 35 weeks gestation sustained a bullet wound in the left lower abdomen with wound of entry situated just above the anterior superior iliac spine and measuring 2 cms in diameter. There was no wound of exit. On physical examination the woman was in mild distress with stable vital signs - pulse 100/min regular ; Respiration 25/min ; BP: 120/80 mmhg and temperature 370^{c}. The abdomen was soft with mild tenderness at the entry wound. Bowel sounds were normal. The uterus was soft, non-tender and symmetrically enlarged with the fundus about 35cms above the symphysis pubis. Fetal heart sounds were present and the rate was 130/min. Per-vaginum examination revealed a closed and unaffected cervix with no blood or amniotic fluid present in the vagina. The presentation of the fetus was cephalic.

A Foley's catheter was passed per urethra and urine found to be clear. Laboratory data: Haematocrit 35% ; Roentgenograams of the abdomen revealed a bullet in the uterus superimposed by the fetal skull (Fig 1.1).

The patient was kept under observation. Eight hours after the injury, the patient started feeling regular uterine contractions. F.H.R. was monitored at regular intervals of 15 minutes and cervix was found to be dilating progressively. The patient delivered a healthy male baby 12 hours after sustaining the injury. Following the delivery, the patient was again examined and was found to be stable. Repeat roentgenogram of the abdomen revealed the bullet in pelvic region. (Fig : 1.2). The patient was observed for one week in the hospital after delivery. She was kept on full orals and was regularly moving her bowels. Being a symptomatic and stable the patient was discharged from the hospital and advised to return for regular follow up's.

However, two months later the patient reported back to the hospital with the same bullet in her hand she had passed the bullet per vaginum during menstruation. The patient was examined again and found to be well maintained. Repeat X-ray of the abdomen showed no bullet in the pelvic region (Fig : 1.3). The bullet was found to be large caliber and intact and was corresponding to the one seen in previous X-rays. (Fig : 1.4).

Discussion:

Penetrating gunshot wounds of the abdomen generally involve multiple organs and patient survival is related to the No. of visceral injuries[3]. The gunshot wounds of the pregnant uterus are associated with a low morbidity and mortality due to following reasons.

I. Uterus itself is not a vital organ

II. Gravud uterus in the later pregnancy occupies a large portion of the peritoneal cavity thereby displacing the small bowel into a more confined space in the upper abdomen and acting as a protective shield to the other important viscera.

III. The bullet striking the uterus (and the conceptus) loses much of its energy resulting in diminished ability to penetrate other organs.

Only 19% of the women with gunshot wounds of the pregnant uterus sustain associated visceral injuries in gunshot wounds of the gravid uterus explains, in part, the excellent maternal outcome : no maternal deaths have been reported in gunshot wounds of the pregnant uterus since 1912 (2).

In case -1, the patient sustained the gunshot wound from a military weapon of large caliber and the bullet had come to rest subcutaneously in the RIF. The bullet had caused associated visceral injury in the form of multiple perforations in the distal jejunum for which resection and end-to end anastomoses was performed. The infant had sustained intrauterine injury and was stillborn. In nearly 60% of cases of gunshot wounds of the pregnant uterus, the infant sustains some injury in utero and perinantal mortality is 41% (2).

The early routine exploration of the penetrating wounds of the abdomen, even gunshot wounds, has been the treatment of choice. However, a review of the world literature by Buchabaum(1-2) propose that selected patients sustaining gunshot wound of the abdomen during pregnancy can be spared exploration. Shaftan[4] has proposed following criteria for conservative management of selected patients.

1. Entrance wound below the fundus of the uterus.

2. The bullet seen radiographically in the uterus.

3. Dead fetus.

In case-I, we think that the exploration was indicated as there were definite signs of peritonitis and the patient did not fulfill the Shaftan' criteria cited above. The wound of entrance was above the fundus of the uterus and immediate radiography was not possible, and the bullet had lodged subcutaneously in the right iliacfossa after traverse the uterus. Moreover, the operative findings of multiple perforation in the small gut justified surgical exploration.

In case-II the wound of entry was in the lower abdomen, below the uterine fundus and the bullet was seen radiographically in the uterus. The patient thus fulfilled the Shaftans' criteria except that the fetus was viable. However, since there was no evidence of any fetal = distress and the patient was haemodynamically stable with no bladder or bowel injury, a conservative trial was prefered.

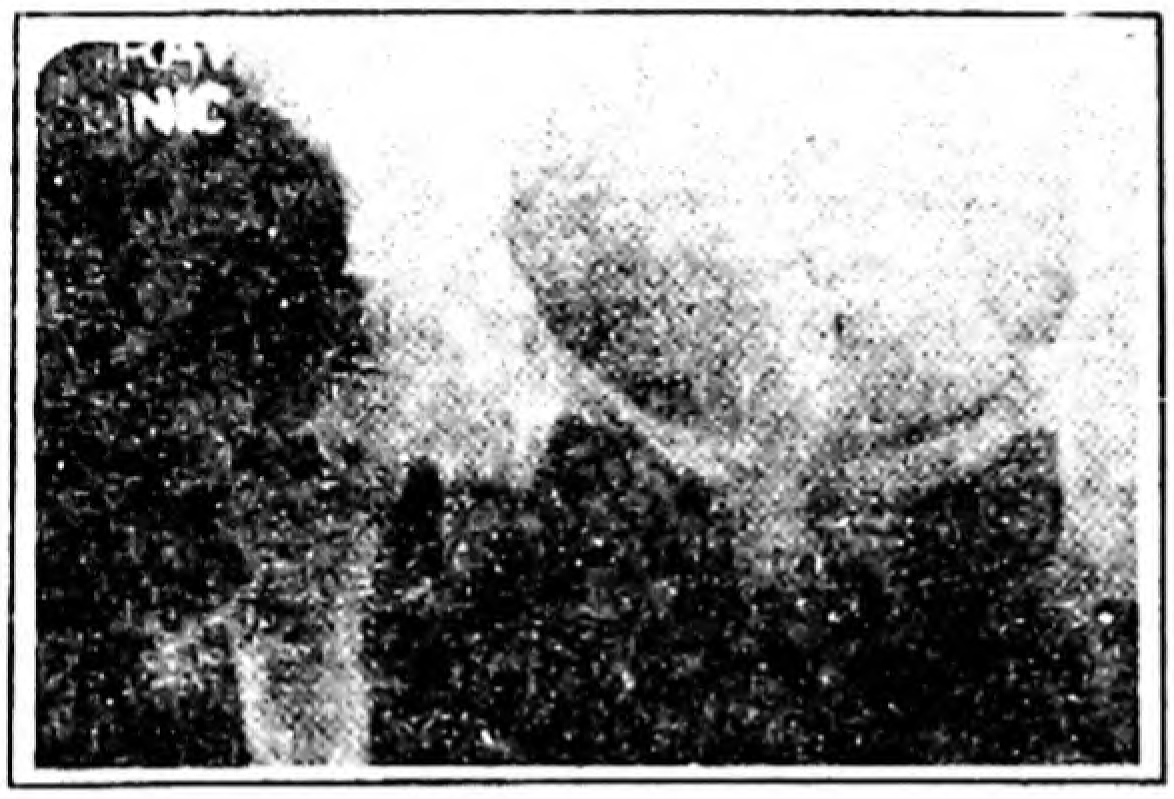

Fig.1.1 X-Ray pelvis showing a bullet with super imposed foetal skull.

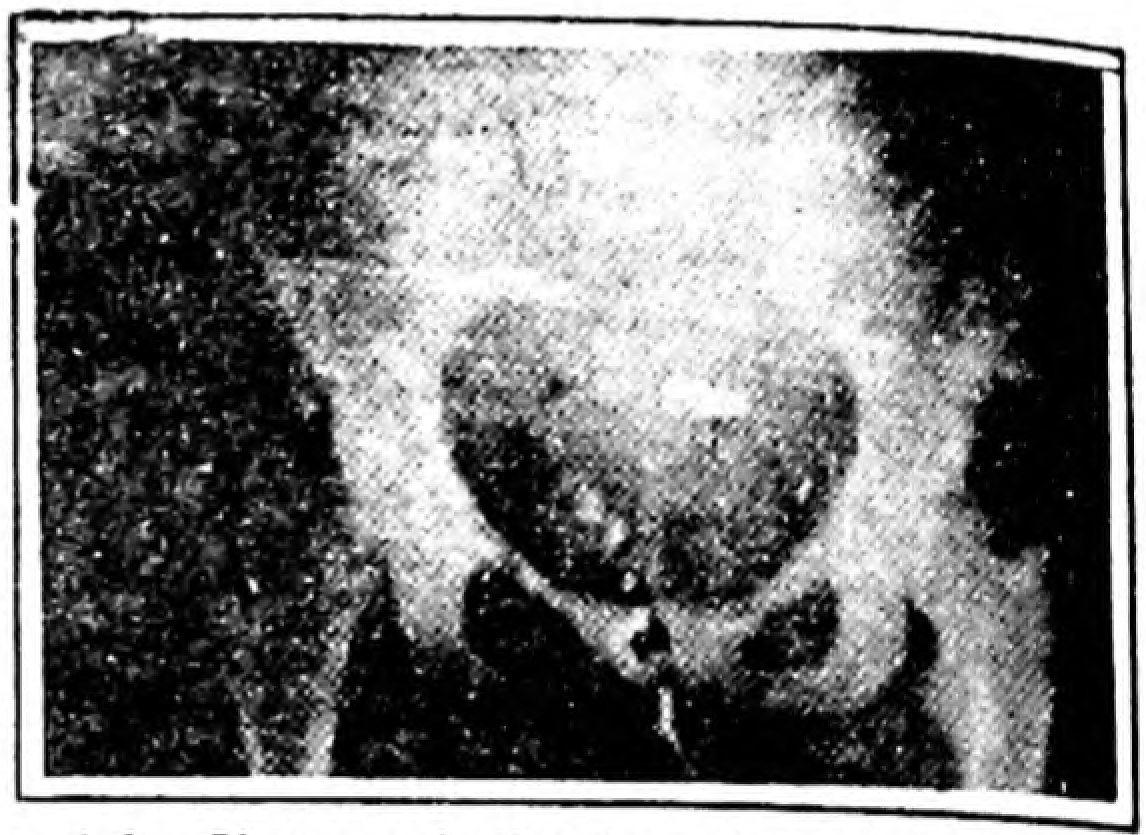

Fig. 1.2 Shows a bullet in pelvis after delevedry of foetus.

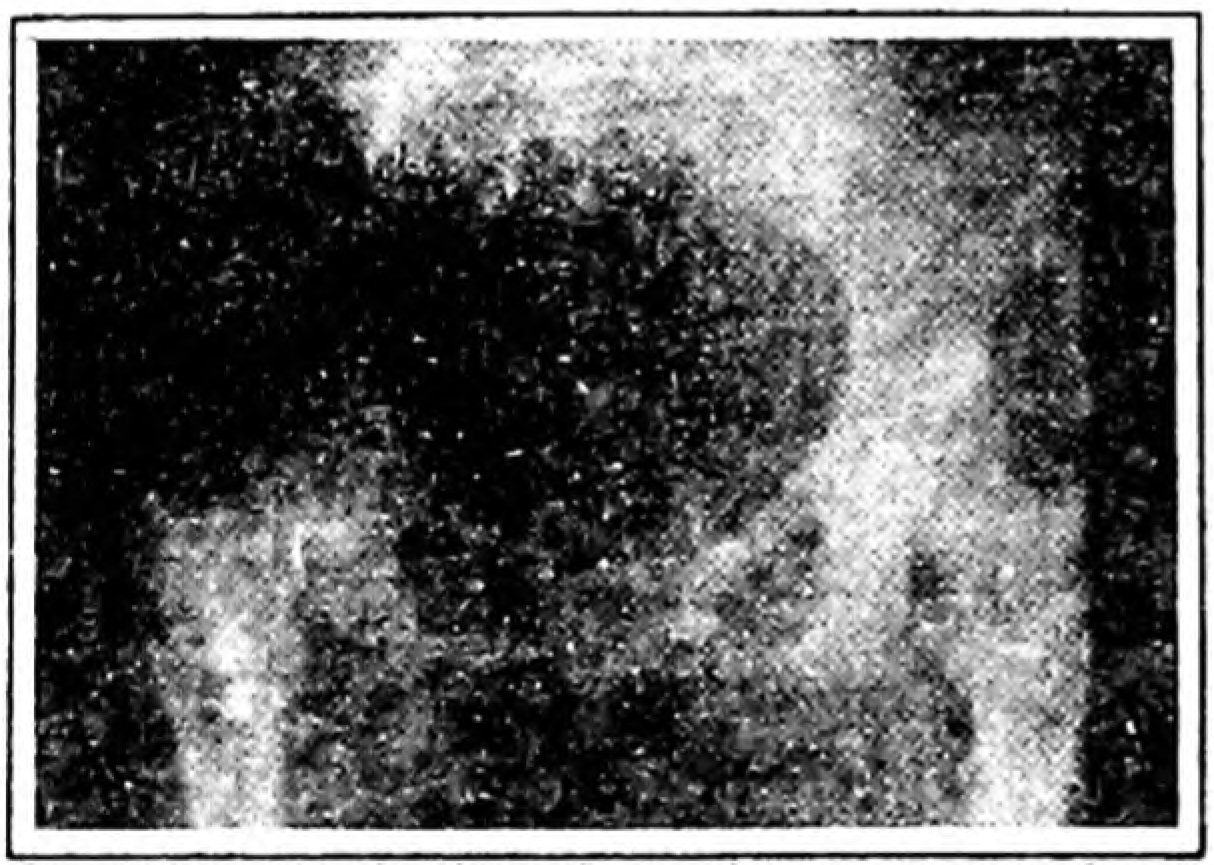

Fig.1.3 No bullet (Passed spontaneously)

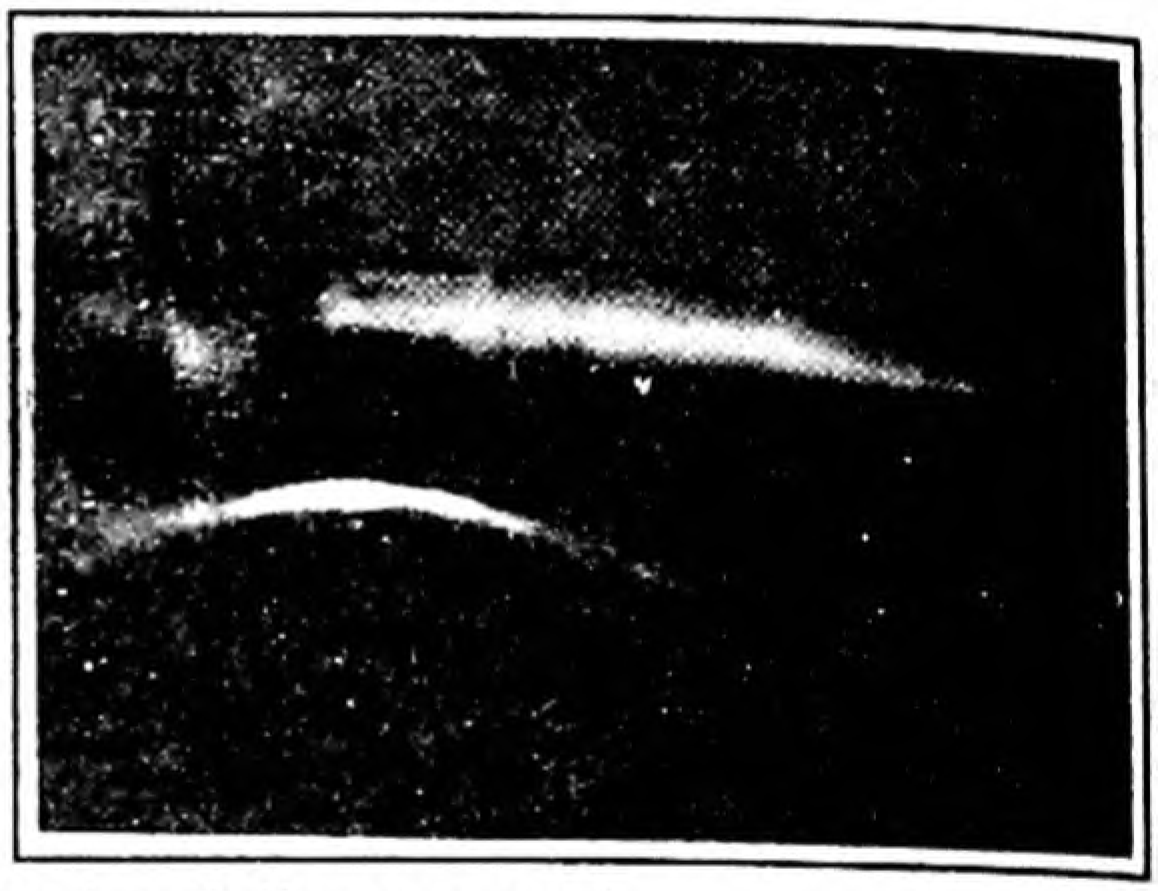

Fig 1.4 Bullet recovered

Reprint Request: Dr. Mufti Mahmood Ahmed 472, C. Jawaharnagar Srinagar Kashmir.

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THYROID ABSCESS PRESENTING AS AIR-FLUID LEVEL IN THE NECK

NISAR A. WANI*, G.H. BHAT*, MUMTAZ-UD-DIN*, M.Y WANI*

Introduction

The occurrence, of thyroid abscess in rare. The relative resistance of thyroid to bacterial infections is well known. A similar case of 70 year old male is presented who had a long standing multinodular goiter and the superimposition is suppurative thyroiditis forced the patient to seek medical attention. Rapid improvement was ensured and complete disappearance of thyroid swelling was observed after incision and drainage of the abscess. Skiagram of the neck showing a huge soft tissue shadow with air fluid level clinched, the pre-operative diagnosis of thyroid abscess.

Case Report:

A 70 years old male hailing from the high altitude mountainous region of Ladakh (J&K) presented to surgical services of S.M.H.S Hospital Srinagar, with a history of recent painful increase in the size of neck swelling associated with fever, rigors and chills. There was history of mild cyspnoe and dysphagia. There was history of swelling in the neck for last 20 years. There was no history Of trauma. No hoarseness of voice, No palpitations, no heat or cold intolerance. Patient was febrile ill looking with pulse 110/minutes, B.P 140/90 mmHg. No cervical lymphadenopathy and no other abnormality was detected on physical examination. Local examination revealed a huge tender swelling 15*12 cms, infront of neck with an area of redness and fluctuations in the centre with raised local temperature restricted movements on deglutition with stretched overlying skin. Laboratory investigations revealed hemoglobin of 12 gms, leukocytosis of 12,000/cmm with predominant polymorphonuclear leukocytosis. X-ray chest was normal. X-ray neck (soft tissues) revealed an air fluid level in the neck with a huge soft tissue shadow. A clinical diagnosis of thyroid abscess complicating a multinodular goiter was made.

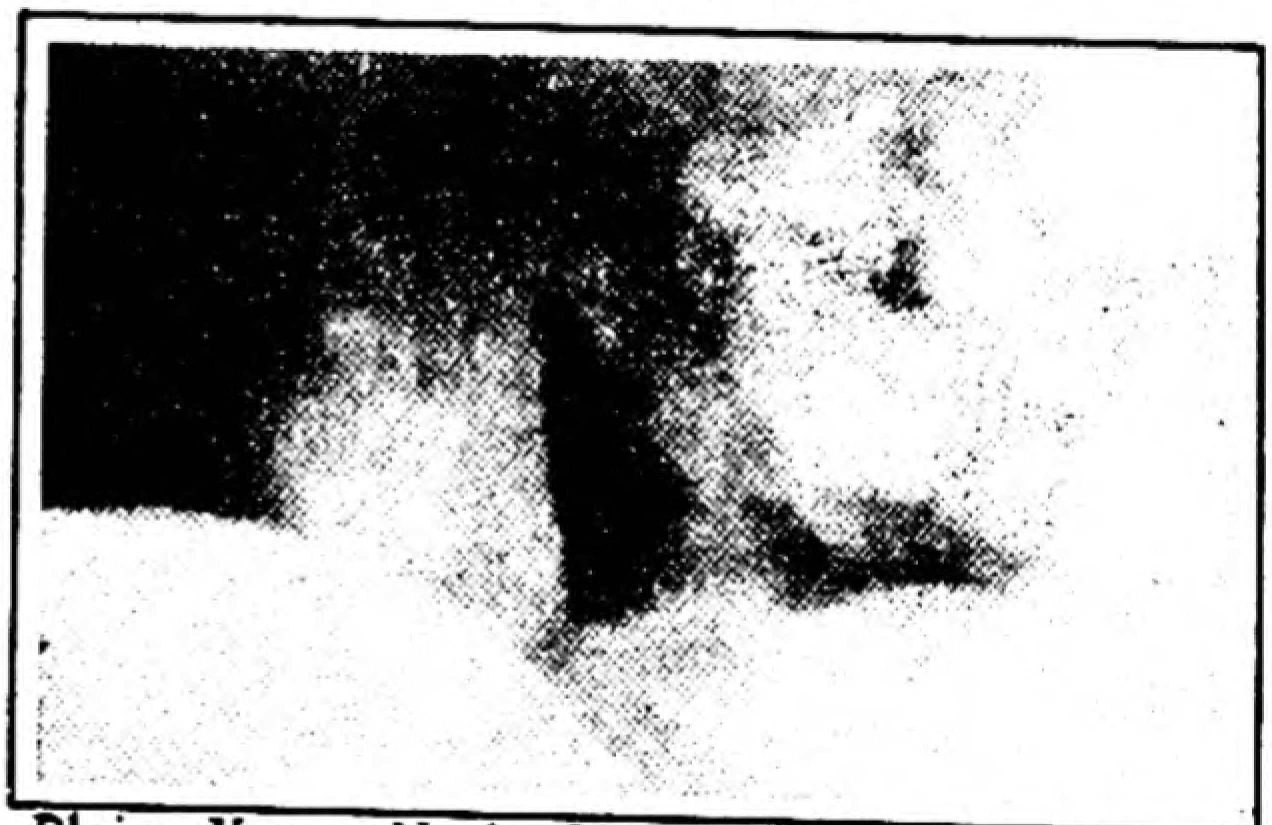
The abscess was incised and drained and about 1½ litre of pus was drained. the cavity was curetted out and histopathological examination of curettings revealed degenerated thyroid tissue with no evidence of malignancy. The swelling gradually subsided and the wound healed well. There was marked improvement in the condition of the patient and he was discharged alright.

Discussion:-

Bacteriathyroidities is a known complication of nocular goiter as reported in the literature (Richie J.L. 1959). A thyroid abscess presenting radiologically as an air fluid level is a rare entity, which has not been reported in the literature.

The relative resistance of the thyroid gland to bacterial infection is well known (Wemach,W.H. 1944). The frequency with which bacterial thyroiditis proceeds to abscess formation is rare. Hypotheses to account for this resistance are (i) Generous vascular and lymphatic supply of thyroid (ii) well developed fibrous capsule (iii) high local iodine level (Schweitzer V.C. and Olson N.R.) Infection of the thyroid gland results from direct extension, blood borne, lymphatic spread or through a patent thyroglosal duct (Hazard J.B. 1955). The recognition of suppurative thyroiditis is important in order to differentiate it from other fascial abscesses of the neck (Charles,H.Man 1977).

The peculiar presentation of thyroid abscess in the old age complicating a multinodular goiter and the X-ray of neck showing a huge soft tissue shadow with a very well marked air and fluid level had been the characteristic features in this case, as these have not been reported earlier in the literature.



Plain X-ray Neck Showing Thyroid Abscess

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OBITUARIES

The faculty of Medical College, the members of Medical College Teachers Association and Editorial Committee paid their condolences in the memory of the following Doctors/ Teachers who died during 1994.

1. Prof. Gh. Rasool (FRCS)
He was an eminent surgeon, teacher, able and strong administrator. He worked for many years as Professor, Head of Surgery G.M College and Medical Superintendent S.M.H.S Hospital Srinagar till 1973. He expired on 6th Nov.94 at Srinagar. He will be remembered for his excellent services in history of Kashmir.

2. Dr. (Col.) Gh. Nabi
Late Col. Gh. Nabi was born in 1912 and graduated from Prestigious King Edward Medical College, Lohore, after that he served in the State forces and the Army. He had a distinction of working in the 2nd World War here he was posted in Central Asia. During his important assignments, he commanded various Field Ambulance Hospitals and 92-GH, Srinagar. He was administrator of S.M.H.S Hospital, Srinagar from 1965 to 1968. He also worked as Superintendent, University Hospital of Aligarh Muslim University. He died on 19th of October, 1994.

3. Dr. Jagdish Singh (MS)
He was an intelligent teacher with full of humor. He served in the Deptt. of Anatomy G.M.College since 1962 and then transferred as HOD. Anatomy Jammu. After retirement he became HOD. Anatomy J.V College Srinagar. He died at Jammu in Dec. 1994 after a short illness.

4. Dr. M. Ashraf Sofi (MD)
A young Doctor, (40 years) hailing from Ganderbal, worked at SKIMS as lecturer Anesthesia, then went Riyad Military Hospital and finally left to U.K. in 1993. He died in U.K. in October 1994 after a short illness.

LABORATORY TESTS OF TYHYROID FUNCTION

"A PRACTICAL APPROACH"

S.T. Hussain*

In treatment of disorders of thyroid function the clinical diagnosis must be confirmed by laboratory tests not only for confirming the diagnosis but also for assessing the response of the patient to treatment. This is especially important as the clinical picture can change markedly during treatment. As a corollory to the above statement it must be emphasized that as the upper and lower limits of normal levels of thyroid harmones vary from individual to individual any laboratory evidence of thyroid dysfunction must be correlated with the clinical findings. The clinical Bio-chemistry laboratory of the department of Bio-chemistry at present performs total T_3, T_4 a d TSH estimations for all patients referred to the laboratory for a small fee which is subsisised by the S.M.H.S. Hospital. For an intelligent use of the hormone estimations by the clinician for the evoluation of the thyroid status of an individual an understanding of the physiology and Bio-chemistry of the secretion of the thyroid hormones and their metabolism by peripheral tissues is essential.

Metabolism of Thyroid Hormones:-

Lobules of the thyroid gland consist of follicles that are a single layer of epithelial cells surrounding a cavity or follicle lumen. The follicular cells take up iodide from the circulation by active transport and then utilise this iodide to iodinate tryosine residues in thysoglobulin a protein secreted by follicular cells in the lumen. Coupling of iodinated tyrosines within thysoglobulin results in formations of T_4 (35%) and T_3 (5-8%).

$$\text{Iodine}\ \xrightarrow[1]{\text{UPTAKE follicular cells}}\ \xrightarrow[2]{\text{ORGANIFICATION}}\ T_4 \,\&\, T_3$$

The first step i.e. uptake of iodide is inhibited by thiocyanate and perchlorate. The second step by carbimazole, propythiouracil and related drugs.

Secretion:- On stimulation TSH is released from pitituary. TSH blinds to receptors and activates all steps of throid hormone systhesis. The thyroglobulin containing colloid is then taken up by the follicular cells and hydrolysed to release T_4 & T_3.

Transport and Metabolisum of T_4 & T_3 by Peripheral Tissues:

In plasma T_3 & T_4 are bound to thyroxine binding proteins namely (1) Thyroxine binding globulin (TBG) and (2) Thyroxine binding prealbumin. Peripheral tissues especially liver and Kidney convert 80% of circulating T_4 to more active T_3 and inactive reverse T_3 (r T_3). Drugs e.g. phenytoin which induce hepatic enzymes increase the rate of this conversion so that plasma T_4 levels fall. In stressed and chronically ill patients and after propranolol administration more of inactive r T_3 is produced. This can be useful as it reduces B M R and energy consumption.

High levels of circulating estrogen e.g. in pregnancy and in women taking oral contraceptives increase Thyroxine binding globulin and so increase blood levels of T_3 and T_4.

A small amount <1% of Thyroid hormones circulates in the free unbound form. Free T_3 and T_4 are the metabolically active fractions (T_3 being 5 times more active than T_4), the Thyroid function tests measure the total (mostly bound) thyroid hormones. Despite this the concentration of the metabolically active free fractions closely parallels the concentration of the bound fraction provided the level of binding protein remains the same. Hence measurement of the total hormone levels can asess the thyroid status of a patient if care is taken to discount changes due to variation in binding protein levels e.g. in pregnancy.

Control of Thyroid Hormone Secretion:-

TSH secretion from the pitituary is inhibited by circulating free thyroid hormones especially free T_4. TSH secretion is stimulated by TRH (Throtropin release hormone) but this can be prevented by excess free T_4. Hence in hyperthyroidism TRH injection does not cause an increase in TRH level and this is used as a test for hyperthyroidism.

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*Deptt. of Bio-Chemistry Govt. Medical College, Srinagar.

Normal Levels:

T_4 3.9 - 10.6 ug/100 ml(50-137 n mol/l)

T_3 0.65 - 1.82 ng/ml(1-2.8 n mol/l)

TSH 0.25 - 5 m U/l

Methods for quantitative measurement of T_3, T_4 and TSH:

Total T_4, T_3 and TSH are estimated quantitatively in serum using solid phase Enzyme Linked Immunoassay (ELISA). Total T_3 & T_4 are assayed by competitive ELISA. After liberating the hormones from binding proteins by treatment with 8 Anilino - 1 Napthalene Sulfonic acid and Sodium salicylate, T_3/T_4 to which the enzyme peroxidase is attached is added and the mixture incubated in tubes coated with antibody. The labelled T_3/T_4 and the serum T_3/T_4 complete for binding to the antibody. After binding, the tubes are washed and incubated with substrate. The T_3/T_4 labelled with peroxidase oxidises the substrate to an orange yellow product. The higher the amount of T_3/T_4 in the serum are less of the labelled T_3/T_4 will be able to bind to antibody and the colour produced on incubation with substrate will be proportionally less permitting quantitative estimation. This is illustreated in the diagram. (Fig 1).

TSH is measured by sandwich technique using two monoclonal antibodies. Tubes coated with anti-TSH are incubated with serum. TSH is bound with anti-TSH. Anti-TSH labelled with enzyme peroxidase is also added. It binds to the TSH bound to anti-TSH. After washing substrate is added which is oxidised to orange yellow product by peroxidase. This is shown in the diagram. (Fig 2).

Factors affecting Total T_3 & T_4:

As mentioned earlier total T_3 & T_4 levels parallel free T_3 & T_4 levels provided the level of the binding protein is not increased and hence can be used for assessing the thyroid function of an individual. However, there may be increase in level of binding protein which could be ministerpreted as hyper or hypothyroidism if only T_4 is measured.

An increase in TBG occurs in

(a) Pregnancy
(b) Newborns
(c) During oestrogen therapy
(d) Use of oral contraceptives
(e) Genetic causes

A decrease in TBG occurs in

(a) Severely ill patients
(b) Nephrotic syndrome
(c) Therapy with androgens or danazole
(d) Genetic causes

To avoid misinterpretation of total T_3 and T_4 levels the above factors must be kept in mind and in cases where altered TBG levels are suspected the results of the tests should be asessed along with the clinical picture to elucidate the true thyroid status.

CLINICAL USE OF THYROID FUNCTION TESTS:

(A) Hyperthyroidism:-

Hyperthyroidism may be a result of a variety of causes. Some causes of hyperthyroidism are

(1) Graves disease
(2) Toxic multinodular goitre or adenoma (Single nodule)
(3) Therapy with Thyroid hormones or rarely iodine
(4) Secretion by tumors
(5) Struma ovarii (thyroid tissue in ovarian teratoma)
(6) TSH secreting tumor of pitituary (very rare)
(7) Early states of inflammatory thyroid disease e.g. Subacute thyroiditis, Hashimotos disease etc. In later stages there is hypothyroidism. In Hyperthyroidism secretion of thyroid hormones does not depend on TSH from pitituary except in TSH secreting tumors of pitituary which are very rare.

INVESTIGATION OF SUSPECTED HYPERTHYROIDISM:

(1) Measure plasma T_4 High levels in a clinically thyrotoxic patient (without suspected alteration in TBG level) confirms diagnosis.

(2) Measure plasma T_3. In T_3 Thyrotoxicosis only T_3 is elevated and so T_3 must be measured before excluding hyperthyroidism.

(3) If T_3/T_4 are in the upper limit of normal but patient shows clinical signs of

thyrotoxicosis it is possible that levels are high for that particular patient. Measure TSH. Low TSH level shows Hyperthyroidism.

(4) Perform a TRH Test. A normal rise in plasma TSH after administration of TRH rules out hyperthyroidism. In this test 200 ug of TRH in 2 ml saline are given i.v. Blood samples are taken at 20 and 60 min. intervals. In normal subjects TSH increases by 2 mu/l & maximum response occurs at 20 min.

(B) HYPOTHYROIDISM:

Hypothyroidism may be primary or secondary.

The causes of primary hypothyroidism are

(1) Hashimotos disease (In early stages there may be hyperthyroidism).

(2) Infalmmatory diseases of thyroid gland e.g. subacute Thyroiditism (In early stages there may be Hyperthyroidism).

(3) Thyroidectomy

(4) Idiopathic

(5) Drugs e.g. lithium

(6) Inherited defects in biosynthesis of thyroid hormones (Dyshormogenesis)

The causes of secondary hypothyroidism are

(1) Anterior pitiutary deficiency in secretion of TSH

(2) Hypothalamic deficiency in secretion of TRH

INVESTIGATION OF HYPOTHYROIDISM:

(1) Measure TSH. A raised TSH level may be the only finding in primary hypothyroidism when results of T_3 & T_4 may be in the low normal range. TSH levels are low in secondary hypothyroidism.

(2) Measure T_4 level. A very low T_4 indicates hypothyroidism unless there is TBG deficiency.

(3) Perrform a TRH test. A subnormal rise confirms secondary hypothyroidism. A normal or exaggerated rise indicates secondary hypothyroidism due to hypopthalamic dysfunction.

Note:- Any physician who wants to do a TRH test must arrange to obtain TRH injection on his own as the same is not available with the Biochemistry Department of Govt. Medical College, Srinagar.

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HIGH LIGHTS OF 1994

Release of the JK-Practitioner (12 NOV.-1994)
Left to Right:
M. Syed (Principal JVC Sgr), G.Q. Allaqaband (Principal, GMC Sgr),

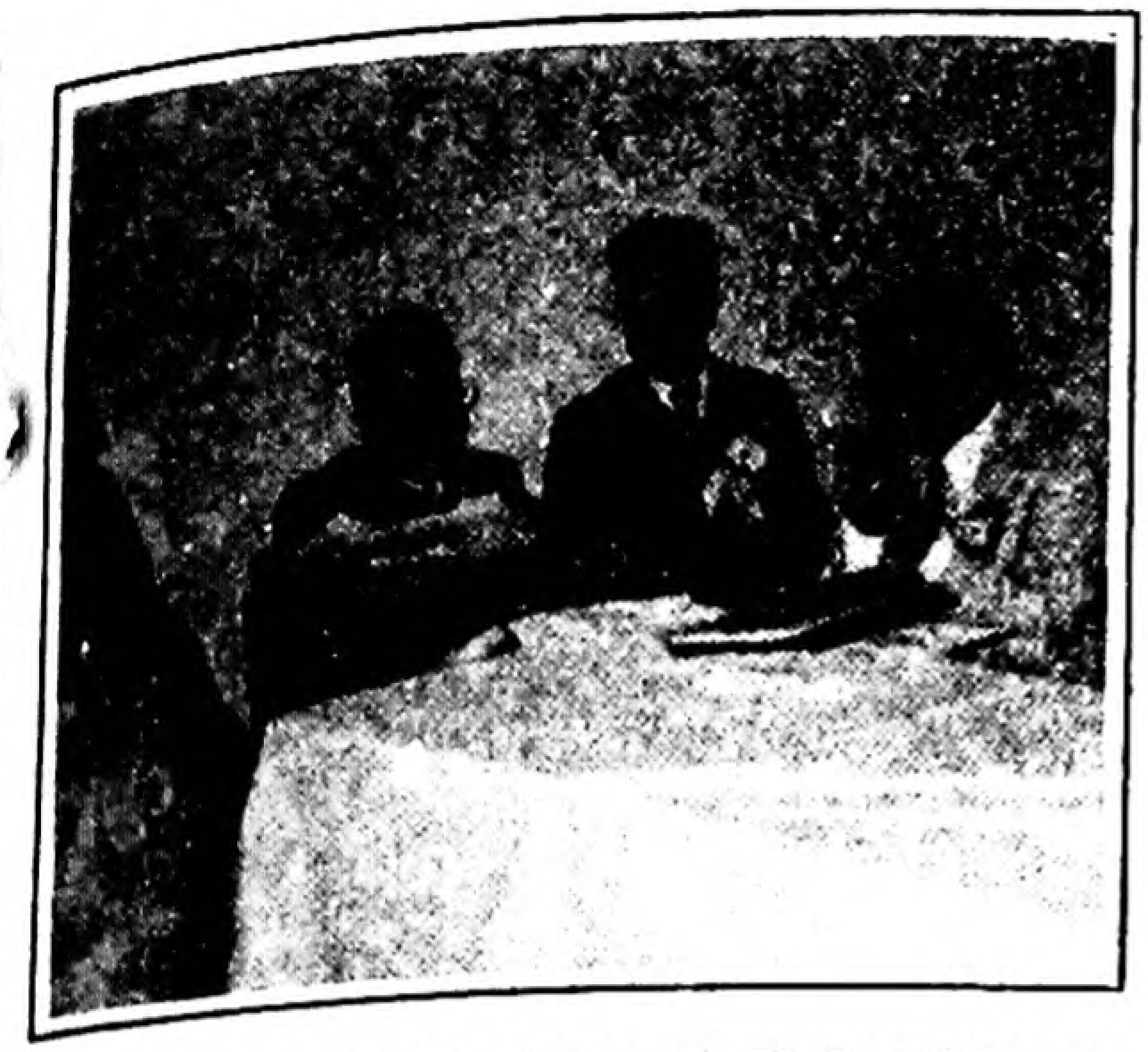
S.Z. Ahmad (Ex. Director SKIMS), G.M. Malik (Editor)
Noor-ud-in (Student's section)

Principal G.Q Allaqaband calls it a Historic Day

Prof. A. Ahad (President, Medical College Teachers Association, appeals for contribution & cooperation from all teachers.

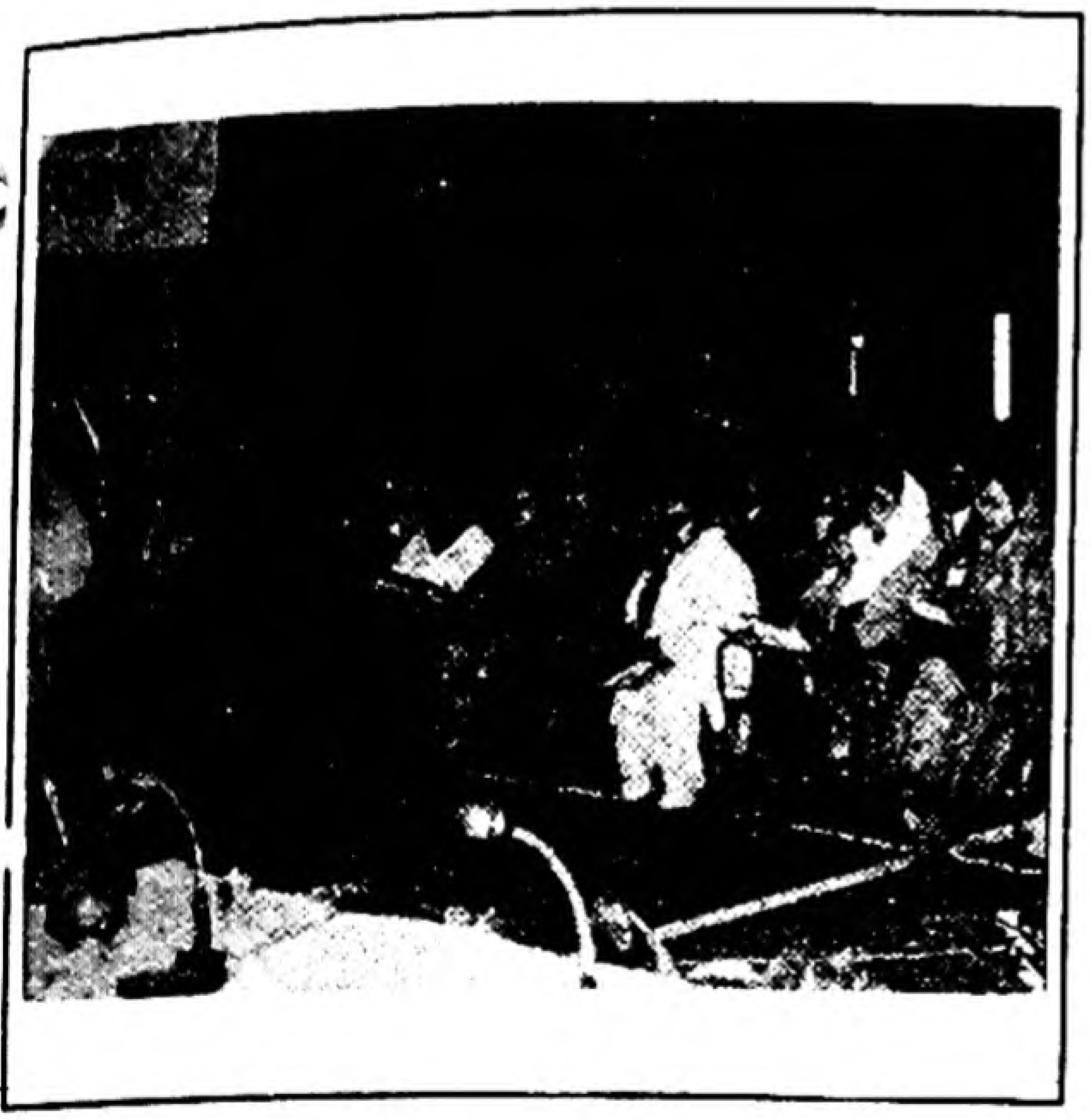
Welcome speach by Editor.

HIGH LIGHTS OF 1994

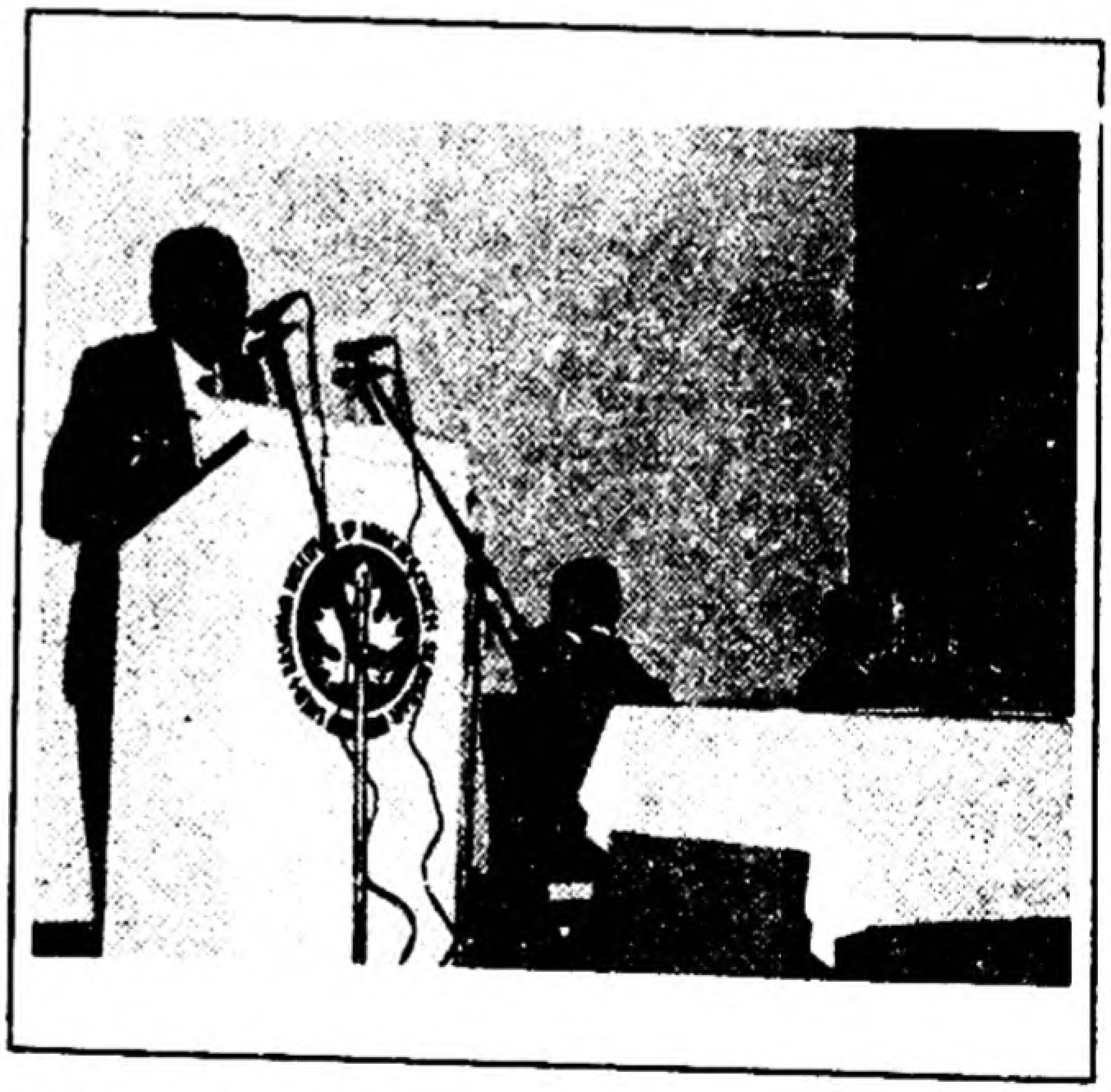


Editor presenting Ist. issue of JK-Practitioner to Professor M.Y Qadri (Vice Chancellor Kashmir University). Professor M.S Khuroo (Director, S.K I.M.S Sgr. (Right side)

Farewell party to Professor G.Q Allaqaband (Principal) by Medical College Teacher's Association and Faculty Members.



Prof. M.S Khuroo (Director S.K.I.M.S) speaks in the Seminar on (CME) "Recent Trends in G.I.T" at Centure Hotel Sgr. December, 1994.



Panel Disscussion on "Maternity and Child Health at S.K.I.M.S Sgr.
From left to right:
Prof. M.S. Khuroo, Prof. G.M Dar,
Prof. G.Q Allaqaband, Prof. Masood
G.M Mattoo (Deptt. of Community Medicine Pediatrics S.K.I.M.S Srinagar)

REHABILITATION OF CANCER DISEASE

BASHIR A. BUTH, (MD., D. PHYS. MED)*

Rehabilitation has become a necessary part of comprehensive care for cancer patients, because of the extent and variety of their treatment related and disease related disabilities.

The majority of cancer patients are in their years of greatest productivity, family, social and community responsibility and therefore are a group of great importance. Rehabilitation of the cancer patient has offered a great challenge in direct ratio to the increasing number of cured and controlled patients. Nearly 40 percent of today's new cancer patients will have a cure and 60% with persistent, recurrent or metastatic cancer will have increased survival periods and 15% of this latter group will still be alive 15 years after the first diagnosis.

Since the time of the survival is increasing due to improved methods of control of the disease, both cured and controlled patients have frequent need for rehabilitation to improve the quality of survival for each individual, so that they will be able to live as independent and productive life, as possible at minimum level of dependency, regardless of life expectancy.

The first step in provision of rehabilitation care is the prompt recognition of the presence of existing disability and ideally the recognition and evaluation of the patient and the provision of the first orders for rehabilitation care can be made at the bed side without waiting to reach a status of readiness for being moved to a rehabilitation service department.

Rehabilitation therapy started early can eliminate development of periods of hopelessness, frustration and despair in cancer patients.

BACK GROUND

Rehabilitation of disability resulting from cancer or its treatment has not been practised until recently. Cancer patients have not been considered eligible regardless of whether their condition was cured, controlled or advanced.

The diagnosis of cancer is associated with fear of the unknown and carries a stigma of dread and an out of proportion sense of finality. The diagnosis of other disease with lethal potential like coronary occlusion and stroke, is usually accepted with far less emotional reaction. Vast rehabilitation efforts are directed towards stroke and heart patients in comparison to cancer patients. This deep seated public understanding of the actual potential that exists for cure or long term survival and associated rehabilitation now possible.

Today's physicians are oriented towards acute disease, and consequently have little interest in disability or its rehabilitation. No cohesive interaction has been developed between various disciplines to assume a role in the comprehensive care of patients to ensure the quality of their survival.

Physicians must be aware of the need for rehabilitation and understand that a proper survival pattern for all patients can provide economic and human returns from the investment in rehabilitation efforts.

MEANING OF REHABILITATION

involves treatment and training of the patient to the end that he may attain his maximal potential for normal living physically, psychologically, socially and vocationally.

There are wide varieties of specific disability found is cancer patients. The problem may be regional related to the site of the disease, or generalized. Brain, spinal cord, or peripheral nerve involvement may cause muscular weakness, sensory loss or paralysis, and require specific care. Peripheral nerve involvement require splinting or support for wrist or foot drop. Patients who may have developed hemiplegia and paraplegia need standard care

---

*Professor and Head, Physical Medicine & Rehabilitation
S.K.I.M.S, Srinagar, Kashmir

programme. Head and neck surgery frequently produce cosmetic problems. Mastectomy may be emotionally depleting and leave physical deficit. The patients undergoing thoracic surgery, need training and assistance in post-operative respiratory functions. Patients who undergo amputation need training and guidance for a suitable prosthesis. Other disabilities which need attention are joint contractures, osteoporosis pathological fractures, urinary calculi, phlebothrombosis and pneumonia.

When disease or its treatment leave the patient with severe physical impairment, a resultant need is created for an adjustment to a new way of life. The sudden knowledge of catastrophic illness can find the individual psychologically ill, unprepared for any change in capacities and status. Simple survival becomes no longer the patient's only cause of anxiety. Attention turns to his or her residual physical limitations and unless specific rehabilitation assistance is immediately available, dependency, depression and fear may become source of deeper problems. Experience has indicated that rehabilitation should begin at the earliest possible opportunity, and be continued through out the entire convalescence, untill maximum benefits can be achieved.

GOALS AND OBJECTIVES OF REHABILITATIONS

The initial goals of rehabilitation of cancer patients are elimination, reduction or alleviation of disability, while the ultimate goal is re-establishment of the patient as a functional individual in his or here environment.

The goals should be selected for each patient after initial evaluation at the onset of care in order to establish a programme that can provide realistic results. Individual goals depend on the certain factors relevant to the patient, and are as follows:-

1. Preventive: When the disability can be predicted, and the appropriate prior training can reduce the severity of its effect.

2. Restorative: if the patient can be expected only to have minimum residual handicap and return to a fully active life style and work.

3. Supportive: when the patient will have to tolerate ongoing disease or persistent disability but can make appropriate gains, towards control of problems, and improved day to day performance. 4. Palliative: if advanced disease and basic disability exist, that cannot be corrected, but where training can aid comfort, performance, and emotional support. This is also important if pain is to be reduced, for the correction of hygienic problems, treatment and prevention independent function, the patient can assure for remaining period of his life.

Other factors to be consider for the programme include age of the patient, type and stage of the disease, other unrelated disabilities present, physical control, social and vocational back ground and basic education. Patient's home circumstance and surroundings must also be considered.

The treating physician should provide initial assessment on which appropriates rehabilitation programme can be based. Planning can then be started early for patient's rehabilitation needs, or referring the patient to the rehabilitation area is likely to be more effective, the sooner such rehabilitation efforts are started.

This early approach is important because bed rest alone allows nearly 3% per day loss of combined muscle power and endurance, and can carry a patient beyond an independent ability to get our of bed. This is also complicated by emotional disability that can result from prolonged inactivity of any kind. The immunized patient should have a suitable physical and occupational therapy bed programme prescribed and instructed.

The meet the goals, the following objectives should be consider:

a)Pd General objectives

1. To provide rehabilitation care for the greatest variety of needs, so as to minimize disability, dependency, emotional stress, and the complications of illness or treatment.

2. To contribute to discharge planning and future purpose of individual patient, as need.

b) Specific objectives (According to individual Goal)

1. To provide preventive measures so as to improve function and reduce morbidity and disability.

2. To provide restorative measure for patients with potential cure of cancer, whose residual disability can be appropriately controlled, or eliminated.

3. To provide supportive measure for patients who must continue with cancer, but can expect relative control or remission for appreciable time, and in whom disability, emotional stress or discomfort can be lessened by rehabilitation care.

4. To provide palliative measure for patients whose disease is advanced and relentlessly progressive, but whose disability, discomfort, and stress can be mitigated by rehabilitation.

The Psychological problems faced by patients, are sometimes as devastating as the physical consequences of disability, therefore attention should be given to psychosocial as well as physical and vocational rehabilitation. Prompt rehabilitations attention can reduce the disability and the time needed for recovery of the patient's handicap.

CONCLUSION

1. Disability from cancer or its treatment can be consider by the same criteria as are used for non-cancer disabilities.

2. It i essential to establish early recognition of patients existing and potential disability and of rehabilitation needs and to stimulate prompt referral for rehabilitation care.

3. Rehabilitation therapy started early can reduce the disability and time needed for recovery of patient's handicap.

4. Each patient should be considered on individual basis, with evaluation of the medical findings, the prognosis, and maximum eventual gain to the patient, his family, and the community, then comprehensive medical care be provided to patient, for improvement in his quality of survival.

5. Realistic goals are necessary for rehabilitation of the cancer patient. The patients should be assisted in reaching improved performance levels. Even in instances where the patients life expectancy is limited, efforts towards maintaining function at maximum potential may produce results that transcend an economic return.

6. The physician should commence intensive medical care and early rehabilitation of the handicapped in acute hospital setting, incorporating rehabilitation services into ongoing programme, is therefore part of comprehensive care for all cancer disabled patients.

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2. Howard, A. Rusk, Rehabilitation Medicine, 4th Ed. 1977.
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CANCER FACTS FOR WOMEN

BREAST CANCER: THE FACTS:

-One of the leading cancer killers of women
-About 46,000 die yearly.
-More likely to happen in women.
· Over age 50
. Who have other family members with breast cancer.
. Who never had children.
· Who were over 30 when they had their first child.

THE GOOD NEWS

-Nearly 9 out of 10 women could survive if:

STAY ALERT

-Check breast monthly
-Age 20-40: Have a health care professional check your breasts every 3 years
-Age 40: Have your first mammogram (breast X-ray)
-Age 40-50: Have a health care professional check your breast every year. Have a mammogram every year or two (ask how often)
-Over age 50: Have a health care professional check your breast every year. Have a mammogram every year.

SEE YOUR HEALTH CARE PROVIDER IF:

-You find a lump in your breast
-You notice any other changes in your breasts

LUNG CANCER

THE FACT

-Is the leading cancer killer of women
-Approximately 59,000 women die each year
-Cigarette smokers are much more likely to get it

THE GOOD NEWS

-More than 40,000 women wouldn't have to die of lung cancer each year-if they just didn't smoke
-If you quit early enough, most damage from smoking will heal

STAY ALERT

-Don't smoke-No cigarette is safe
-Can't quit yet? Smoke less, especially low tar and nicotine cigarettes
-Don't use tobacco products (chewing tobacco, snuff)
-Stay away from people who are smoking

SEE YOUR HEALTH CARE PROVIDER IF:

-You have a nagging or painful cough that doesn't go away
-You cough up blood
-Your chest hurts all the time

UTERINE CANCER (CERVICAL/ENDOMETRIAL)

THE FACTS

-Approximately 46,000 new cases a year
-Can strike women at any age

THE GOOD NEWS

-Fever women die from cancer of the uterus each year-because they have regular pap tests and pelvic examinations.
-The pap test detects signs of cervical cancer early, usually in time to treat it

STAY ALERT

-Have a pap test every year of you are:
Over 18
·Sexually active
-You are more likely to get endometrial cancer if you are: Over 40, overweight, taking hormones, have abnormal bleeding, are infertile, or do not ovulate

SEE YOUR HEALTH CARE PROVIDER IF:

-You have any abnormal bleeding
-You have reached menopause and are overweight to taking hormones

COLON AND RECTUM CANCER

THE FACTS

-About 74,000 new cases a year
-Third major cancer killer
-Most often strikes women over age 50

THE GOOD NEWS

-Finding out early can save more than 4 out of 5 people

STAY ALERT

-Most likely women:
·Have ulcerative colitis (a painful condition Your health care professional can diagnose)
·Have a family member with colon or rectum cancer
·Have a family member with polyps in the colon of rectum

·Over age 40: Have a digital rectal exam each year

·Over age 50: Ask a health care professional what kind of regular exams you need

SEE YOUR HEALTH CARE PROVIDER IF:

-You bleed from the rectum

-You see blood in your stool

-You see a persistent change in your bowel movements

-You have cramping pain in your abdomen

SKIN CANCER

THE FACTS

-Women with light skin are more likely to get it

-About 2,600 women die each year from melanoma, a serious skin cancer.

THE GOOD NEWS

Most skin cancer can be cured

STAY ALERT

-Avoid mid-day sun and too much exposure to the sun

-When you or your family member are outside, make sure to cover up exposed skin

·Wear clothing that covers head, arms, neck, and legs

-Use a "sun blocker" lotion oı creme (SPF 15)

SEE YOUR HEALTH CARE PROVIDED IF:

-A birthmark, mole, or other spot on your skin starts to change

WOMEN AND CANCER

Too many women die each other from cancer. Cancer of the breast, lung, uterus, colon or rectum are the most common cancers that attack women.

Protect your self by reading the information in this booklet. Keep a copy where you can look at it if you have any questions. Early detection (finding out right away that a cancer is growing) gives you the best chance to be cured.

A health care professional can answer many questions and give you any test you need. If you're between age 20 and 40, have yourself checked for cancer once every three years. Over 40, get checked every year.

SEE YOUR HEALTH CARE PROVIDER RIGHT AWAY IF YOU HAVE:

·A sore that doesn't get better.

·A nagging cough, or unusually hoarse voice.

·Indigestion (very bad upset stomach more than once in a while) or problems with swallowing.

·Changes in a wart or mole.

·Unusual bleeding or discharge.

Thick spot or lump in breast or anywhere else.

·Change in bowel or bladder habits.

CANCER:

WHAT CAN YOU DO TO PROTECT YOURSELF?

There are no guarantees, but there is hope. The American Cancer Society knows a lot about what seems to cause cancer. They also know that finding cancer early makes treatment easier and more effective. Thousands of people each year are "cured" of cancer. Better yet, thousands more are learning how to be "cancer smart". Reading this chapter is one way to be "cancer smart". Read it carefully, and keep it where you can read it again later. When you have a question, no matter how small, ask a health care provider or call the American Cancer Society. Getting answers in one of the best ways to protect yourself!

Source:-
AMERICAN CANCER SOCIETY

HAPPY IDD GREETINGS

TO

OUR READERS AND SUBSCRIBERS

Students page

CURRENT AMERICAN CANCER SOCIETY GUIDELINES FOR THE CANCER-RELATED CHECKUP

Test of Procedure	Sex	Age	Frequency
Sigmoidosocpy preferably flexible	M & F	50 and over	Every 3-5 years
Fecal Occult Blood Test	M & F	50 and over	Every year
Digital Rectal Examination	M & F	40 and over	Every year
Prostate Exam*	M	50 and over	Every year
Pap Test	F	All women who are, or who have been, sexually active, or have reached age 18, should have an annual Pap test and pelvic examinations. After a woman has had three or more consecutive satisfactory normal annual examinations, the Pap test may be performed less frequently at discretion of her physician.	
Pelvic Examination	F	18-40 Over 40	Every 1-3 years with Pap test Every year
Endomerial Tissue Sample	F	At menopause if at high risk**	At menopause and thereafter at the discretion of the physician
Breast Self-Examination	F	20 and over	Evert month
Breast Clinical Examination	F	20-40 Over 40	Every 3 years Every year
Mammography***	F	40-49 50 and over	Every 1-2 years Every year
Health Counseling and Cancer Checkup****	M & F M & F	Over 20 Over 40	Every 3 years Every year

* Annual digital rectal examination and prostate specific antigen should be performed on men 50 years and older. If either is abnormal, further evaluation should be considered.

** History of infertility, obseity, failure to ovulate, abnormal uterine bleeding, or unopposed estrogen or tamoxifen therapy.

*** Screening mammography should begin by age 40.

**** To include examination for Cancers of the thyroid, testicles, ovaries, lymph nodes, oral region and skin

Source: American Cancer Society.

MEET THE PROFESSOR:

FARROQUE AHMAD KHAN, M.B.
Chairman, ·Department of Medicine,
Nassau County Medical Centre
Professor of Medicine,
State University of New York
Stony Brook.

Graduated: University of Kashmir, 1965
Born: February 1943 (SRG)

Postgraduate Training: Medicine/Pulmonary: Queens Hospital Center; Jamaica, NY Albert Einstein College of Medicine; Bronx,NY

Board Certification: Medicine - 1972, Pulmonary - 1972, Critical Care - 1987

Medical School Activities:
Professor of Medicine: 1985-Present; Excellence-in-Teaching Award:1980; Distinguished Faculty Award: 1981;Elected to *AOA Mu* Chapter: 1989; Department of Medicine Executive Committee: 1981; - Present: Queens Hospital Center:
Chief, Division of Pulmonary Medicine: 1977 - 1986

Nassau County Medical Center:
Chairman, Department of Medicine: 1987-Present Director, Internal Medicine Program with a total of 99 house officers

Research Interests: Respiratory failure, granulomatous lung diseases, Pulmonary infections, medical education, health reform.

Publications: Over 100 publications dealing with critical care, infections, granulomatous diseases, medical education, etc.

Presentations: Ninety presentations at national and international conferences, on subjects dealing with various aspects of Pulmonary medicine.

Special Programs: Since 1973, organized and moderated 25 special teaching programs in the U.S.A. and abroad, including six biennial conferences on advances in pulmonary medicine, co-sponsored by The American College of Physicians. Member, International Medicine Education Committee of the Association of Program Directors in Internal Medicine:1990 - Present.

Visiting Professor: University of Southern California: 1980, 1989; University of Kuwait: 1982, 1983, 1989; harvard Medical School: 1982; University of Kashmir: 1983, 1985, 1989; Jordan Medical School: 1985, 1986; University of Kentucky-Lexington: 1986; Cleveland Clinic: 1982, 1984, 1987; University of south Carolina: 1988; University of Auckland, New Zealand and Sydney, Australia: 1992.

American College of Physicians:

Organized six biennial conferences on advances in cardiopulmonary medicine in 1980, 1982, 1984, 1986, 1987, 1988.

Meet-the-Professor session and workshops on mechanical ventilation at the annual session of The American College of Physicians in 1988 - 1994.

Research Grants:

Twenty-nine grants totalling over 2 million dollars from National Institutes of Health. World Health Organization. Pharmaceutical agencies, voluntary organizations, ets. for support of various investigative, educational and treatment protocols.

Awards

Was awarded *Mastership* by The American College of Physicians in 1994.

Books

In collaboration with several program directors from upstate and downstate New York, prepared and edited a book which will be resource manual used for house staff training. Scheduled for publication in 1994.

CONFERENCE IN SRINAGAR

Dr. Farrouque Khan with the cooperation of many ACCP Indian Chapter Officers was principal organizer of a highly successful meeting on Cardiopulmonary Medicine which met in Srinagar, Kashmir in August 1985

Standing L to R: Z. Ahmad (FRCP), G.Q. Alaqaband (FRCP), F. Pirzada (MD, FCCP) R. Bono (MD,FCCP) M. Ahmad (MD,FCCP), F. Khan (MD,FCCP), A. Jan (MRCP), S.Jalaludin (MD,DM), H.Rissam (MD)
Seated L to R: K.J.Kazi (MD,FCCP), G.M.Malik (MD,FACG), M.Sajjad (MD,CCP), and G.Kawoosa (MD,FCCP)

Published in:BULLETIN VOLUME 8, 1985.

Dr. Farroque Ahmad Khan
(FRCP,MB,MACP)

Clincical Images

Case Note:- A 37 years female presented with loss of appetite and a firm Mass in Epigastrium since 7 months. Clinical diagonosis-? N-P Stomach. Investigation:-

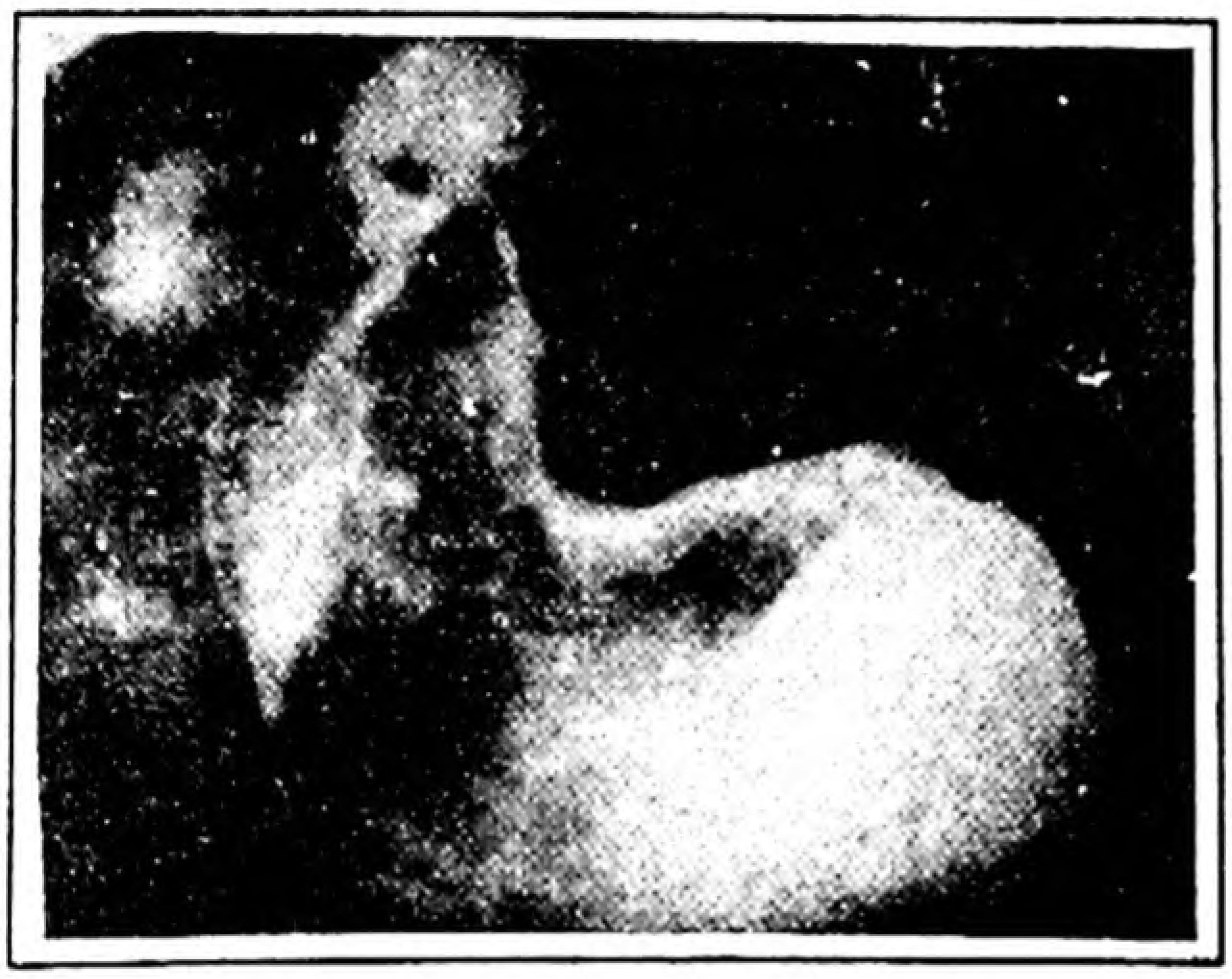

Fig. 1. Barium X-ray showing filling defect with streaks of Barium going inside.

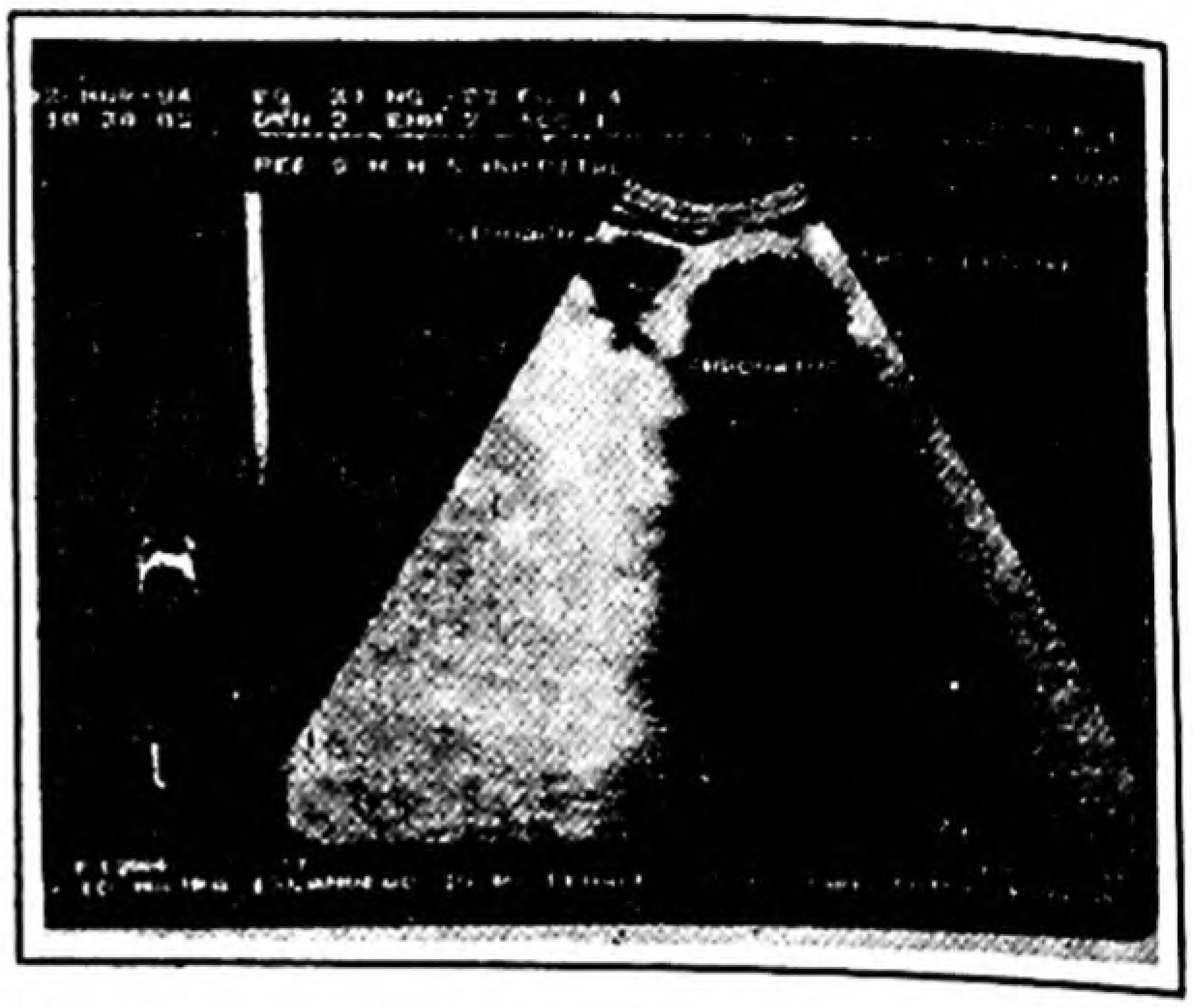

Fig. 2 Ultrasound showing calcified mass with shadowing.

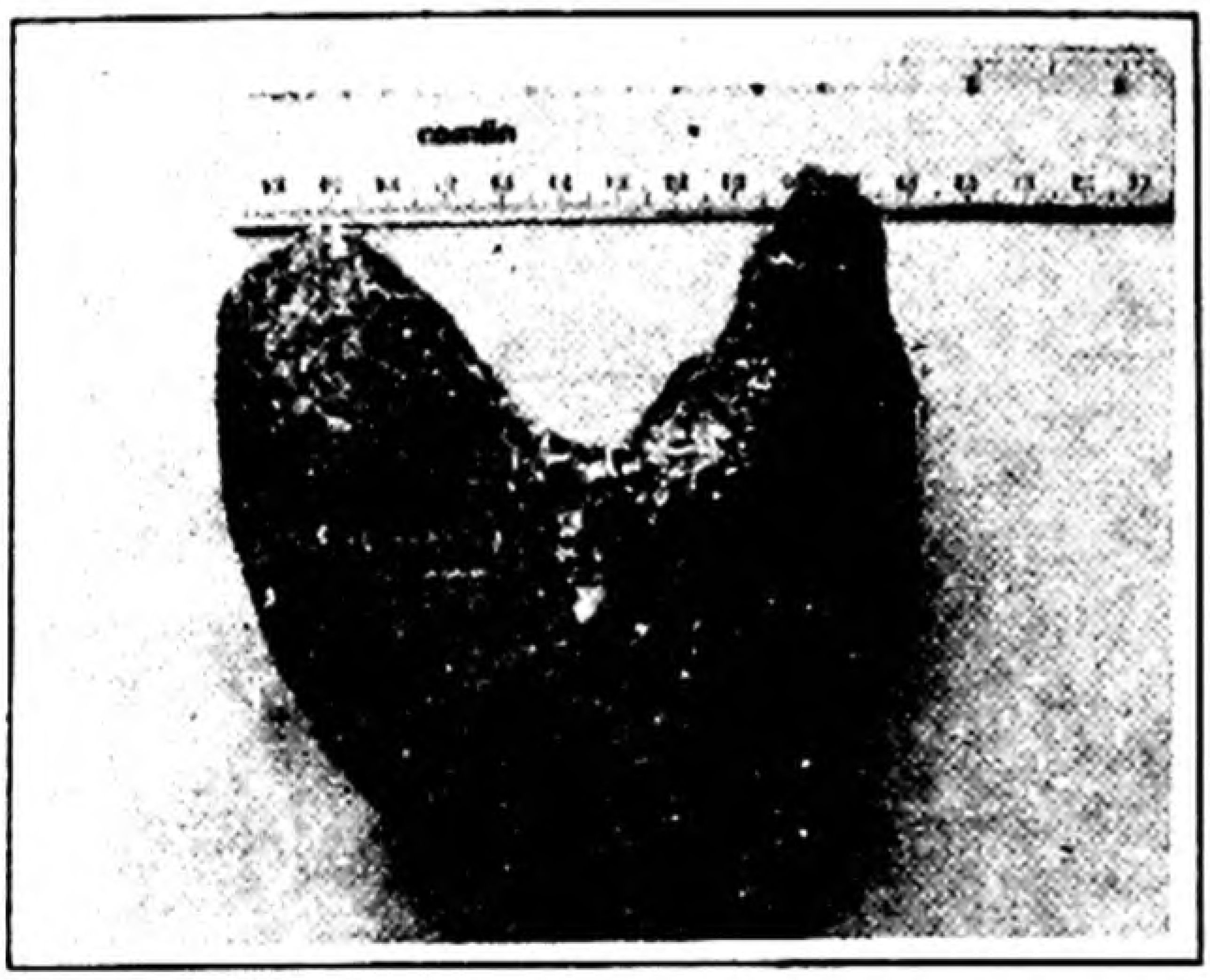

Fig. 3 Gross speciman showing Hair Ball (Removed by surgery)

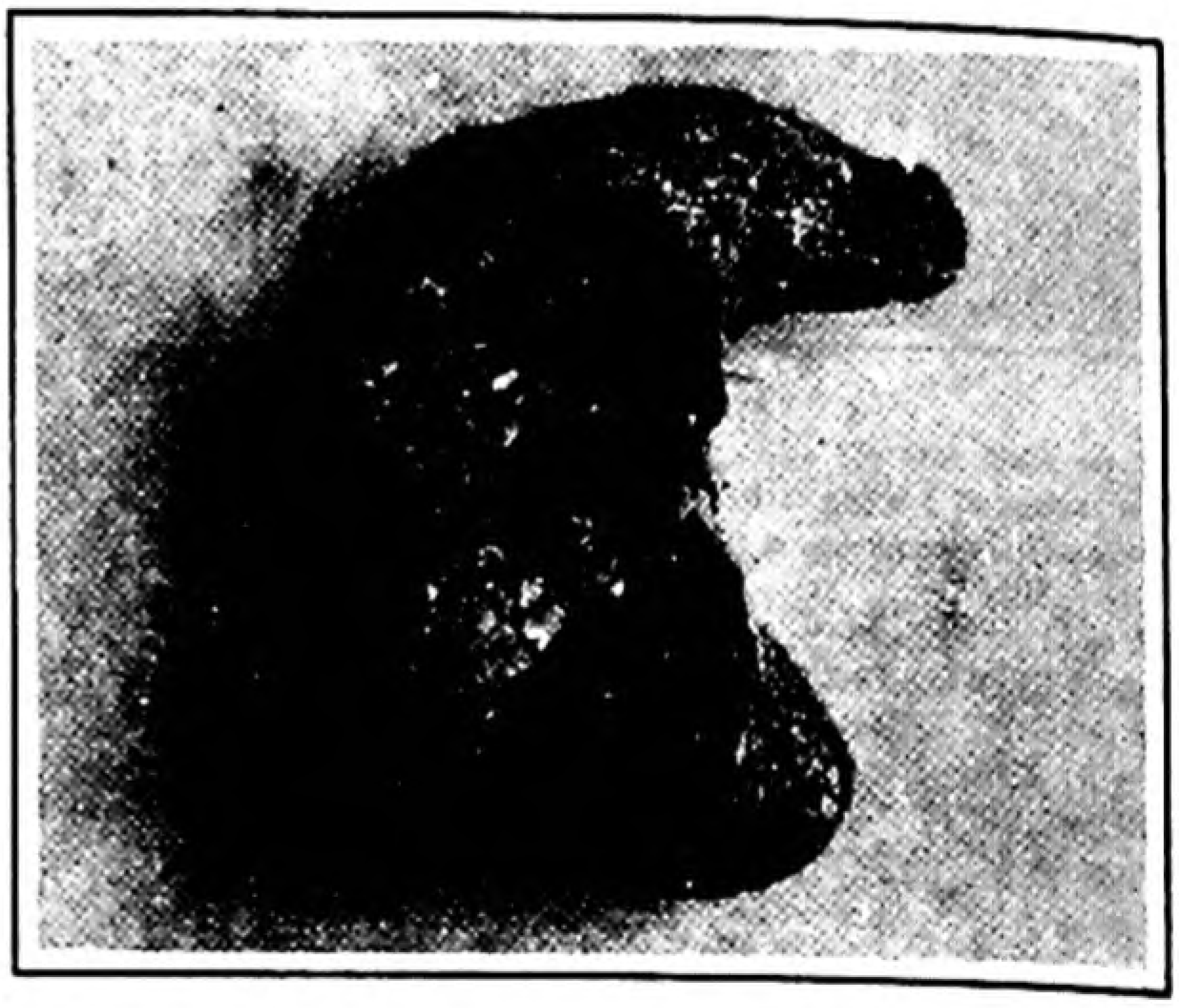

Fig. 4 Transverse showing rice fruit skin and Hair.

Final diagonosis:- Trichophytobezoar

Authors:- G.M.Malik (MD., FACG), M. S. Malik (MS), Mubarik (MD), S.Kadla (MD), Naveed (MD.
(Deptt. of G.I.T., Surgery and Radiology Medical College, Srinagar)

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FIGHT SIX KILLER DISEASES
IMMUNIZE YOUR CHILDREN

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AGE GROUP	B.C.G.	D.P.T	POLIO	MEASLES	D.T.
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2½ Month	-	2nd Dose	2nd Dose	-	-
3½ Month	-	3rd Dose	3rd Dose	-	-
9 Month	-	-	-	One Inj.	-
16-24 Months	-	Booster Dose	Booster	- Dose	-
5 Years	-	-	-	-	One Inj.

SAVE MOTHERS FROM UNTIMELY DEATH

Give Pregnant Ladies :-

(1) Inj. Tetanus Toxoid as early as possible.
(2) Inj. Tetanus Toxoid Booster Dose-one month after first Dose.

ENSURE GOOD HEALTH

Arrest Water Borne Diseases:-

(1) Use safe boiled chlorinated drinking water.
(2) Promote hand washing with soap and water.
(3) Use Iodized salt for prevention of GOITER.

LEPROSY & TUBERCULOSIS ARE CURABLE

(1) Report the nearest Health Institution in case any patch especially if loss of sensation is noticed on the body of exclude Leprosy.
(2) In case of constant cough for more than 15 days with fever, loss of appetite and loss of weight., Report to the nearest Health Institution to exclude Tuberculosis.
(3) AIDS is Preventable. Contact local health authorities.

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nerve endings in the skin which may inhibit melanogenesis and could have a toxic effect on melanocytes. Autonomic dysfunction in the vitiligenous patches in the form of vasoconstriction and augmented sweat response have been observed [13] suggesting an increased adrenergic activity at the lesional nerve endings. Ultrastructural studies have indicated anatomic contact between nerve fibers and melanocytes [14]. Cases of segmental vitiligo are common [6], in which the lesions are in dermatomal distribution. To support neurogenic hypothesis,however,no chemical agent has been demonstrated by researchers at the nerve endings with the requisite destructive effect on the melanocytes [6].

d) Melanocyte self-destruction hypothesis

Lerner [2] suggested that melanocytes destroy themselves due to a defect of natural protective mechanism that removes toxic melanin precursors. Tyrosine, DOPA and dopachrome are cytotoxic to the melanocytes [15]. However, these precursors of melanin pigment do not attain toxic levels in vitiligenous skin. In vitiligo depigmentation is more common in those areas that normally are more pigmented and these are the sites of accelerated melanogenesis [2]. The melanocyte self destruction hypothesis is based on the clinical features of vitiligo and on experimental studies of cutaneous depigmentation by chemical compounds that have a selective lethal effect on functional melanocyte [16].

Pathology

The essential process in vitiligo is the destruction of melanocytes. Long standing lesions of vitiligo reveal no melanocytes on staining with silver or dopa reaction. Early lesions and the peripheral areas of enlarging lesions that are hypopigmented rather than depigmented show dopa positive melanocytes and some melanin granules in the basal layer. At the border of the lesion melanocytes often appear large and possess long dendritic processes filled with melanin granules [17]. Some authors have described presence of a dermal infiltrate at the border of depigmented area. Regarding Langerhan's cells (suprabasal clear cells) some observers have noted an increase in their number in vitiligenous epidermis while others have noted no increase in the total number of Langerhan's cells but a shift towards the basal cell layer [17]. Mishima [18] suggested a dynamic inter-relationship between melanocytes and intermediate cells. According to this hypothesis indeterminate cells are inactive melanocytes and they increase in proportion to the disappearance of active melanocytes in vitiligo.

Clinical features:

Vitiligo can occur at any age but most of the cases are seen in the active growth period [12,19]. Nearly 50% of all vitiligo cases occur below the age of 20 years and about 70-80 below the age of 30 years [12]. Lowest age is reported to be at birth [6] and highest is of 97 years [20]. Vitiligo affects both sexes equally. Vitiligo, in a predisposed individual is precipitated by severe emotional stress, sunburn, major illness, surgical procedure, pregnancy, parturition and physical trauma [6,12,19].

Vitiligo is characterized by milky white macules of varying size and shape which gradually spread peripherally and frequently exhibit a hyperpigmented border. The texture of the skin is unaltered.

Thereare two types of vitiligo, Type A and Type B,(tableI) [21]

	Type A	Type B
Distribution	Nondermatomal	Dermatomal
Ratio	3	1
Onset	Any age (<20 years in 50%)	Young
Activity	Life long	Rapid spread 1 year.
Association with halonevus	Yes	No
Association with immunologic disorders.	Yes	No

Table-1. Vitiligo (type A & B) clinical manifestations

Vitiligo has also been classified as vitiligo areata, zosteriformis, mucosae, acrofacial (lip tip syndrome) and universals [4]. The initial site of involvement is commonly around the body orifices, over bony prominences, exposed areas, sites of repeated trauma (Kobner's phenomenon), body folds, mucous membranes or the pigmented areas of the body [6]. Grey hair may frequently be observed over the vitiligenous patches (leukotrechia) [6].

Vitiligo has been observed to occur in association with or closely follow certain cutaneous disorders like lichen planus, psoriasis, alopecia areata and localized pruritis [4]. Halonevus may be presenting feature in some cases of vitiligo [6]. Associated systemic disorders include hyperthyroidism and Hashimoto's

·MIXED LINEAGE LEUKEMIA

Based on the concept of lineage fidelity, leukemogenesis represents a clonal expression of hemopoietic cells arrested at discrete stage of differentiation. The use of leukemic cells as models of normal differentiation implies that they are a true reflection of normal development. Some cases of leukemia simultaneously or sequentially express characteristics of more than one linkage and some leukemic lymphoblasts show a certain degree of asynchrony in their antigenic expression compared to the normal hemopoietic counterparts, indicating that leukemic cells may not be true replicas of the normal cells.

Use of reagents to characterize the antigenic expression by normal lymphoid, granulocytic or monocytic subsets has been added by the development of monoclonal antibodies and flow cytometry. The identification of differentiation somatic gene rearrangements has provided means of examining lineage and differentiation stage of hemopoietic cells. A significant percentage of leukemic cells express antigens, gene rearrangements or both of more than one lymphohematopoietic lineage; besides, there is a greater discordance between immunophenotype of leukemic cells and those of normal differentiating cells. Many characteristics (e.g. immunoglobulins, antigens) once called lineage specific are now lineage associated.

Acute leukemias with blasts expressing more than one hematopoietic lineage called hybrid, biphenotypic, biclonic simultaneous, billineal, synchronous, metachronous, chimeric, or mixed and lineage Switch leukemia. Acute leukemias in which individual cells coexpress both myeloid and lymphoid characteristics are called Acute mixed lineage linkage leukemia. For heterogenous leukemia (those in which a single cells displays lymphoid or myeloid features but not both) are called Billineal leukemias. Billineal leukemias can be synchronous (in which there is simultaneous distinct population of leukemic cells of more than one lineage) or metachronous (in which the lineage is expressed following the other). Metachronous leukemia is recurrence of the original leukemic clone with changes in the cellular expression of lineage associated antigen or gene rearrangements associated with another lineage to distinguish the case from treatment induced Second malignancy, the re-appearance of the original clone must be demonstrated by the presence of common cytogenetic abnormality or identical gene rearrangements. Lymphoblasts may express a single myeloid associated characteristic suggesting an aberrant expression or persistence of stem cell associated phenotype.

The diagnosis of mixed lineage leukemia requires morphological, cytochemical, immunological,molecular, Immunological gene rearrangements, T cell receptor (TCR) gene rearrangement study, elucidation of cytogenetic abnormalities (karyotypic characteristics). Each has been assigned a relative weight and given values. A score of 2 or more in each lineage is considered as mixed lineage leukemia. Morphology is unincluded as characteristic and does not indicate lineage reliably, however gene rearrangements are reliable indicators of cell lineage.

Lymphoid criteria	Points
Cytoplasmic Ig	02
Ig LC gene rearrangement	02
Karyotypic abnormalities	02
37°C rossette formation	02
Surface TCR expression (WT 31)	02
Ig-HC gene rearrangement	02
Tdt expression	01
TCR gene rearrangement (gamma/beta)	01
CD 19,20,21	01
CD 22(cytoplasmic and surface expression)	01
CD 3 (cytoplasmic and surface expression)	01
CD 5 or CD 8 expression	01
Myeloid criteria	
MPO (more than 3% by low or E/M)	02
Auer rods	02
ANB	02
Platelet peroxidase	02
Karyotypic abnormalities	02
IO F7,CD 41,CD 42, d expression	02
SBB	01
CD 13,CD 33,CD 14,CD 15,Leuk. 9 expression	01

Table-I - Criteria for the diagnosis of mixed lineage leukemia (2 or more points from each category are required)

Leukemias which express a single marker of another lineage, thus unqualifying as mixed lineage in the proposed system are called either Lymphoid leukemia with myeloid associated antigen expression or Myeloid leukemia with lymphoid associated antigen expression.

Clinical significance of Mixed lineage leukemia:

a. There is decrease in remission rate after standard induction treatment, especially in children.

b. In cases of adult ALL, there was decrease in CR rates with blasts which expressed myeloid associated antigen; however, if at all remission was attained, it was brief.

c. Children ALL (FAB) with myeloblasts (Myeloid antigen positive) did not correlate with the poor prognosis and need not be, to use more intense and aggressive treatment. It is important to distinguish patients who go having true lineage Switch or metachronous expression of lineage characteristic from patients with second malignancies occurring after intense chemotherapy. Reinduction treatment should be directed towards predominant found at the time of relapse. Second CRs are difficult and if obtained, brief.

d/. The clinical significance of asynchronous antigen expression of ALL is unknown. It may imply that the disease is not a prototype of maturation arrest. If the cell surface phenotype does not correlate with the maturational stage of normal lymphoid differentiation, separation of leukemia according to the developmental stage would be untenable. Prognosis of the lymphoid malignancies may be related to the degrees of deviation from the normal than maturational difference. More studies are needed to prove such developmental deviation.

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EDITORIAL

SOCIAL MEDICINE - LESSON FROM KERALA

Kerala with its tremendous social progress has put to envy not only the developing countries but many developed nations as well. This small state, tucked away in the South-west corner of India, represents only 1.18% of the total area of India but sustains 3.43% of its population. With area of 38.863 sq km and a population of 2,90,11,237 (1991) Kerala is the most crowded state of India with a population density of 747 per sq km.

The per capita income of Kerala falls short of the national average (Rs 4220 : Rs 5670; 19991-92), and the daily per capita calorie intake is just 2158 Kcal well below the ICMR recommended daily allowance of 2400 kcal for Indians. Because of historical and climatic compulsions, the state has always preferred development of commercial agriculture more than food crops, and therefore, is 50% short of food. There is 1 doctor for as many as 6470 people as against 1 for 2148 at the national level. While at the all India level, 62.30% households have reasonable access to potable water, in Kerala only 18.89% house have access to safe drinking water.

Historically, Kerala is a young state which didn't exist at the time of independence and came into existence only on Ist November, 1956 when the 2 princely states of Travancore & Cochin, and previously British- administered territory of Malabar were amalgamated. Politically, the State has been a classic example of see-saw politics-there have been 16 changes of Ministry since its first election in 1957.

With this grim scenario, Kerala would seem to be the last state to have achieved the remarkable social development its has so proudly done.

Health & Social Indicators	All India	Kerala
Death rate /1000	9.7	6.0
Birth rate/1000	31.7	19.6
I.M.R.	80	17
Average births per Woman	4	1.9
Annual growth rate (%)	2.2	1.4
Life expectancy at birth	58.6	66.5
Life expectancy (Women)	59	74

Table-I Comparison of Kerala & All India Health Statistics (1993)

It is evident from Table I that Kerala is far ahead of the rest of India with respect to health status of its citizens. Kerala is distinguished for its low child death rates alongwith low fertility. While the nation is committed to a long-term couple protection rate (CPR) of 60% by the year 2000 AD, Kerala had already moved ahead with 80% of its couples using family planning in 1991 (when national average was just 43%)

UNICEF attributes Kerala's social progress to its progressive social policies alongwith its tradition of participatory democracy and strong commitment to female education. Kerala is the only Indian state where women outnumber men (1040:1000), and again the only state where female literacy is nearly universal (87%), when the rate on all India level is as low as 34%. In a country where 50% of girls stop out of school before completing their 5 years of schooling, Kerala has a zero drop out rate among the grades 1-5. There has always been a strong political and social commitment to female education in Kerala. This state has contributed India's first women magistrate, first women Munsiff, first women sessions judge, first woman High court judge, first woman chief engineer and first woman Director of Animal Husbandry. The latest feather is its cap has been the first ever woman to become a member of the country's highest judicial body, the Supreme court. Almost universal female education has raised mean age at marriage from the national average of 18.32 to 21.85 years, partly attributable to increased female employment. It has also helped women to make wise and responsible decisions in matters of marriage, family, children, food and health.

Kerala has demonstrated that adequate health can be attained by poor states (like J&K) provided strong political and social commitment is forthcoming. Our own state is hopelessly lagging behind in all social and health indicators more so in female literacy, In fact, there never has been a strong political commitment to female education. All the odds have been against education of girls especially in the rural areas. Low status of women has been an important factor. Girls have always been regarded as 'belonging to other's and consequently, any investment in their education has been treated as a loss without returns to the parents. Co-education also have been a main deterrent to female education in our highly conservative muslim society. Policy makers could never motivate parents to send their girls to school. Incentives for female education which should have been introduced at the primary level of education were deferred till college education. Employment of rural girls in gainful economic activities has been a very important factor in encouraging parents to withdraw their girls from schools, but at the same time it has helped female children to prove themselves as productive citizens as their male siblings.

As long as money continues to play a pivotal role in our social life and poverty and economic insecurity loom large, we can never dream of motivating rural masses to send their young daughters for schooling which not only is not monetarily productive but instead needs money to sustain even with completely free education in our State. In near future, it is inconceivable that any significant improvement is going to occur in the state of female education, if our usual social apathy continues.

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Endoscopic Management of Esophageal Varices : Current Status

D. K. Bhargava

Bleeding from the esophageal varices is the most common cause of upper gastrointestinal hemorrhage in India [1]. The discouraging results of medical and surgical treatment modalities and the improvement of fiberoptic instruments led to the resurgence of an old technique; endoscopic variceal sclerotherapy (EST) in the 1970's [2]. During 1980's two other procedures, namely endoscopic variceal ligation, [3] and endoscopic clipping [4] came into existence. However, an ideal treatment for variceal bleeding is still not available.

Endoscopic Sclerotherapy (EST)

EST is considered as a relatively new technique though it was first introduced in 1939 by Crafoord and Frenckner. This method has gained acceptance all over the world using fiberoptic endoscopes [5,6].

There are numerous variables that influence the results of EST. These include clinical variables, technique variables and skill of the operator. Clinical variables include differences in patient characteristics, etiology of portal hypertension and severity of underlying liver diseases. The type, quantity and concentration of the sclerosants also influences the results [7]. The usual sclerosants used are polidocanol, sodium tetradecyl sulfate, ethanolamine oleate and alcohol in developing countries. On the basis of controlled studies we found polidocanol to be superior than either alcohol or sodium tetradecyl sulfate [8-11]. Other issues which are more or less settled now include the superiority of intravariceal to paravariceal injection and that one weekly injection is preferred for long term management till varices are eradicated.

EST has proved to be beneficial for control of active variceal bleeding [12-14]. It prevents rebleeding during long term management and also improves survival of the patients. However EST is associated with rebleeding in 25-50% of patients before eradication of varices; variceal obliteration takes 6 to 8 weeks and complications related to the procedure are present in 15-50% of patients [15-17]. The complications are mainly retrosternal chest pain, fever, presence of deep ulceration which may cause bleeding, stricture formation and at times perforation. Pulmonary complications like pneumonia, mediastinitis and pleural effusion are also common. The rate of complication depends upon the type and concentration of sclerosant used. Till today, we do not have an ideal sclerosant. There was no effect of omeprazole or sucralfate in reducing the complications [18]. The optimal technique of sclerotherapy is yet to be standardized.

a. Active variceal bleeding Emergency EST for control of active bleeding is the procedure of choice. It controls bleeding in 72-100% cases. In our experience the procedure is technically feasible in 97% of patients with an initial success rate of 88%. However rebleeding occurred in 18% necessitating repeat EST. Avoidance of balloon tamponade and vasopressin infusion prior to EST have no effect on the technique of injection. It is equally effective in patients with differing etiologies and Child's categories. However rebleeding episodes are higher in patients with cirrhosis of the liver and NCPF than in those with extrahepatic obstruction (EHO). Rebleeding is related to the severity of liver failure, being higher in patients with worsening hepatic functions [13].

b. Long term management EST has been found valuable for the long term management of portal hypertension. This is mainly due its ability to prevent recurrent variceal hemorrhage by eradicating the varices. Our studies on the basis of more than 700 patients indicate that 28% patients of cirrhosis of the liver, 24% with noncirrhotic portal fibrosis (NCPF) and 11% having EHO rebled from varices prior to the eradication. Eradication was achieved in 80% cirrhotics, 88% in patients with NCPF and 92% in patients with EHO group. The mean number of sessions of sclerotherapy required for eradication was 7 irrespective of the etiology of portal hypertension. Recurrence of varices was observed in 16% of patients after a mean period of 31.6 ± 26.9 months and were between Grade I and Grade II at the onset. An analysis of the survival by the Kaplan Meier technique showed

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the probability of survival at 10 years was 88%, 80% and 50% in patients with EHO, NCPF and cirrhosis of the liver [19-21]. Thus the patients having good liver function (EHO & NCPF) had a better response to sclerotherapy than cirrhotics in terms of rebleeding, bleeding free period and survival [22-25].

c. Prophylactic sclerotherapy Prophylactic EST is controversial in general and studies have demonstrated high rebleeding and excessive mortality rates as compared to those treated with medical management alone. Propranolol treatment has advantages over EST especially in Child's A status patients [26,27].

Endoscopic Band Ligation (EVL)

Steigman in 1988 introduced EVL both during active bleeding and for long term management. Using rubber bands, acute variceal bleeding could be controlled in 90% of cases. Eradication of varices was achieved in 80% of cases requiring a mean of 5-6 sessions [3,28]. Subsequently three controlled trials [29-31] comparing EST and EVL favoured endoscopic variceal ligation. EVL controls active bleeding like EST. Variceal obliteration occurred significantly with fewer sessions. The rebleeding prior to eradication of varices and complication rates were also significantly lower in patients treated with EVL than those treated with EST. The technique of EVL has been standardized.

In spite of these advantages there are several drawbacks associated with EVL. It eradicates only larger varices, and smaller varices may not be eradicated. EVL does not obliterate perforating vessels and thus recurrence of varices may be faster than EST and a long term follow-up in terms of recurrence of varices and patient survival, is not available as yet. The other unfavourable factor is repeated reloading and insertion of endoscope and ligator reduces the endoscopic field of vision by at least 30%. To overcome these difficulties overtube is recommended. Overtube has its own disadvantages as it can lead to mucosal tears and subsequently perforation. Other complications are related to O ring itself. These include chest pain, transient dysphagia, shallow and large ulcers and bleeding from the ulcers.

EVL & EST As both the procedures have disadvantages and advantages over each other the concept of combining EVL & EST is appealing. Combination of these techniques eradicates the varices much faster than either alone [32,33]. In a study by the author a combination of EVL & EST eradicated the varices (Grade O) in 76% in contrast to 30% by EVL. In addition 56% of the varices were reduced to grade I. There was no statistical difference in terms of sessions required for eradication and usual complications associated with EST were not present. In the authors experience combined procedure may be more beneficial than either of them separately.

Endoscopic clipping Miyoshi et al [4] have recently reported the use of hemoclips for esophageal varices whose principle is similar to band ligation. They have inserted 3 to 20 clips per case over 1-4 sessions. Small varices were eradicated in 1-2 sessions and larger varices could only be reduced in size. Varices recurred in 3 (33%) of the 9 patients (33%) within a follow-up period of 6-18 months. Major complications were not observed. The authors recommend combining clipping along with less aggressive sclerotherapy to achieve better results. Further studies are needed to prove the efficacy of this procedure.

Conclusions

i. EST is the procedure of choice during active bleeding as technically it is easier to perform than EVL.
ii. For long term management EVL may be superior to EST, though more long term dated are required.
iii. Combined EVL & EST may be promising.

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New Horizons in Endoscopy

Hysteroscopy as Diagonostic aid in Gynecological Diseases

Farida Shawl

Endoscopic techniques have become indispensable to the practice of modern obstetrics and gynecology. Most anatomic structures as well as disease processes of female reproductive tract conveniently lend themselves to this method of examination. Patients are searching alternatives for major operative procedures. Endoscopy provides as direct information and post operative recovery time can be reduced. It utilizes natural passages and requires no incision. New operative techniques will broaden indications and further facilitate the out patient management of intrauterine problems.

The first satisfactory endoscope was used by Desormacex in 1853 for the examination of urethra and bladder, but he also mentioned a possible use in the uterus. However, the first success in hysteroscopy was reported by Pantaleonic in the "Medical press and Circulation" of July 1869.

Instrument:

It may be:

(A) Contact Hysteroscopy with:
  a) Optical guide;
  b) Cylindrical chamber that collects and traps the light;
  c) Magnifying appliances

(B) Panoramic Co_2 Hysteroscopy with :
  Fibre-optic telescope

Anaesthesia:

The services of many multiparous women are patulous enough to allow the insertion of 6 mm hysteroscopewithout need of dilatation. When dilatation is in need, the procedure may be performed under general or paracervical block anaesthesia.

Distension media is used for panoramic hysteroscopy. Most Commonly used media are :

i. Dextran 32% W/V in 10% dextrose
ii. Carbon-dioxide gas
iii. Dextrose 5% in water.

Indications of Hysteroscopy:

1. Abnormal premenopausal and post-meno pausal uterine bleeding.
2. Diagnosis of sub-mucous fibroids or endometrial polyps.
3. Location and of retrieval of lost intrauterine contraceptive devices or other foreign bodies.
4. Evaluation of infertile patients with abnormal hysterograms.
5. Diagnosis and treatment of intrauterine adhesions.
6. Division of small uterine septae.
7. Exploration of the endocervical and uterine lesions with repeated pregnancy losses.
8. Evaluation and staging of adenocarcinoma of the endometrium.

The list of possible indications is long, and technique cannot be considered to be the solution of all problems, rather it is an adjunct to other techniques in the diagnosis and treatment of intrauterine lesions.

In our hospital where majority of the patients are reporting for the above mentioned gynecological diseases and we are using this procedure as yet for diagnostic purposes only and will prove better for diagnostic purposes intrauterine lesions.

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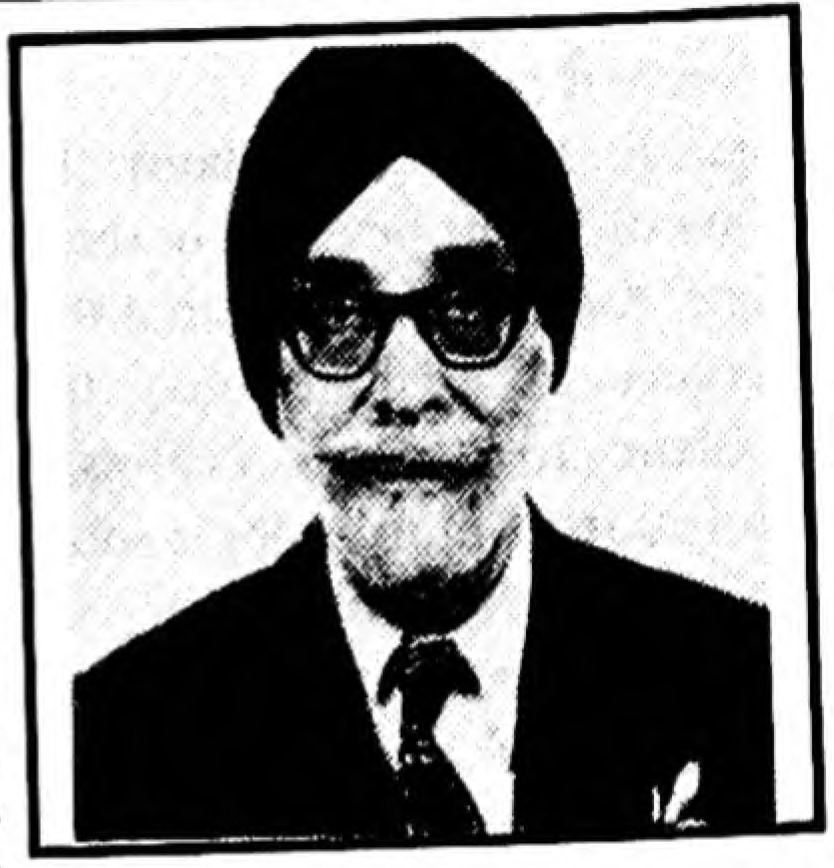
In Memoriam



Late Dr. Harbhajan Singh, FRCS

(1911-1991)

Late Dr. Harbhajan Singh, FRCS was born on 8th August 1911 in village Kanihama, Baramulla District. He had his early education at Kanihama, Baramulla and Srinagar. After passing his FSC, he joined King Edward Medical College Lahore, and completed his MBBS, in 1936. Being one of the top six graduates he was appointed as House Surgeon in the Department of Eye, Ear, Nose and Throat in K.E.Medical College Lahore for period of one year. In 1939 he went to England and did his postgraduation in London obtaining Diplomas in Eye and ENT. He was appointed in Moorfield Hospital London and ENT hospital Gray's Inn Road London as Clerical Assistant till 1941.

He returned to India in 1941 and was appointed as Eye and ENT specialist at Jammu in 1942. In 1949-50 Dr. Harbhajan Singh was selected as Civil Surgeon in Himachal Pradesh. He was persuaded by Sheikh Mohd Abdullah, the then Prime Minister, to return to the state and serve the people. In 1950 he rejoined the Kashmir Medical service. Dr. Harbhajan Singh pioneered Eye and ENT Surgery in the state and was an outstanding surgeon and clinician. He also served as superintendent SMHS Hospital, SMGS Hospital Jammu, Deputy Dean SMHS Hospital and Principal AMT School. He headed the Eye and ENT department Medical college Srinagar with distinction and continued as Professor and Head till his retirement in 1970.

He continued to practice after retirement and was inducted into the council of Ministers by Sheikh Mohd Abdullah in 1977. He held the charge of Minister of state for Health and Medical Education till 1981. He resigned in 1981 and returned to his profession and continued actively practicing till 1990 till ill-health forced him to give it up. He passed away in Dec.1991 after a brief illness. Dr. Harbhajan singh shall be remembered in the valley as a brilliant surgeon, compassionate Doctor, gifted teacher and a humble man with a penchant for serving his fellow countrymen amidst numerous out-of-the-country lucrative pastures. May his soul rest in peace.

Review Article

Vitiligo

Aftab Ahmad, Sheikh Manzoor Ahmad

Vitiligo is an idiopathic acquired depigmentation of skin characterized by ivory-white macules of different size and shapes, which tend to enlarge peripherally in the course of time [1]. Vitiligo is the commonest of all pigmentary disorders and it is stated that 1% of the world population has vitiligo [2]. In India 3% of population is reported to suffer from vitiligo [3]. Vitiligo is a major sociopsychological problem in dark skinned races.

Regarding the nomenclature [4] of the disease, there are different views about the origin of the term "Vitiligo" Bateman believes that the glistening white appearance of the vitiligenous patches bear a striking resemblance to the flesh of calves (Vituli). Fitzpatric also points to its morphologic similarity to the white patches of spotted calves (Latin "vitiligo" means calf). Leider and Rosenblum ascribe the term to the latin word "vitium", which means a blemish or a fault. In India, occasionally vitiligo is referred to as "white Leprosy" (Shweta Kushta or Dhaval Kushta), thus confusing it with leprosy [4].

History

Vitiligo has plagued mankind since antiquity and the earliest authentic reference was made in the ancient book of Hindu mythology, Atharva Veda [5] which dates back to 1400 BC, and in other ancient books like Charak Samhtra (800 BC) and Manu Simriti (200 BC). It was described as "Shweta Kushta" which probably meant vitiligo [4].

Etiopathogenesis

The precise pathogenesis of vitiligo is yet an enigma. Various hypotheses, not mutually exclusive, have been promulgated. Genetic, clinical, physiologic, biochemical, histochemical, immunologic and experimental studies have time and again been undertaken to elucidate this aspect. Of these the most accepted theories include genetic, autoimmune, neurogenic and melanocyte self-destruction hypotheses [4].

a) Genetic theory

Although there are no specific genetic markers for vitiligo [6], the high incidence of family history in various hospital based studies suggest the genetic etiopathogenesis of vitiligo [4]. The occurrence of vitiligenous lesions at the same time and location in monozygotic twins has been documented by Mohr [7]. Finco et al [8] found that in vitiligo, there is significant increase in the frequency of HLA A30, B27, CW6 and DR7; and a significant decrease of HLA DR1 and DR3 as compared with controls. An increased frequency of HLA CW6, B27 and DQW3 was found in pediatric form (younger than 12 years of age) of vitiligo; A30, CW6, and B27, DQW3 in early onset form (younger than 16 years of age); and A30 in the adult form (older than 16 years of age).

b) Autoimmune hypothesis

An autoimmune mechanism has been postulated as pathogenic factor for vitiligo on the basis of its association with a number of disorders which are also considered autoimmune. Incidence of vitiligo in patients with autoimmune disease is 8-15% compared to 1% of general population [8]. Thyroiditis, pernicious anemia, Addison's disease, diabetes mellitus, localized scleroderma, alopecia areata and halo nevus have been observed in vitiligo patients [4]. Antibodies to normal melanocytes have been detected by using immunoprecipitation assay [9]. Haring et al 10] recently using a new enzyme-linked immunosorbant assay (ELISA) to study relationship between the presence of these antibodies and activity of vitiligo demonstrated IgG pigment cell antibodies in 80% of the patients with active vitiligo and their absence in inactive vitiligo controls and concluded that there is a correlation between the incidence and level of pigment cell anti-bodies and disease activity in patients with vitiligo, suggesting that vitiligo is an autoimmune disease mediated by an immune reaction to pigment cells. In a recent study [11], the role of tyrosinase, a key enzyme involved in melanin synthesis, was studied and the results have shown that tyrosinase is a principal autoantigen in vitiligo.

c) Neurogenic hypothesis

This hypothesis was put forth by Larner [12] who suggested that a compound is released at peripheral

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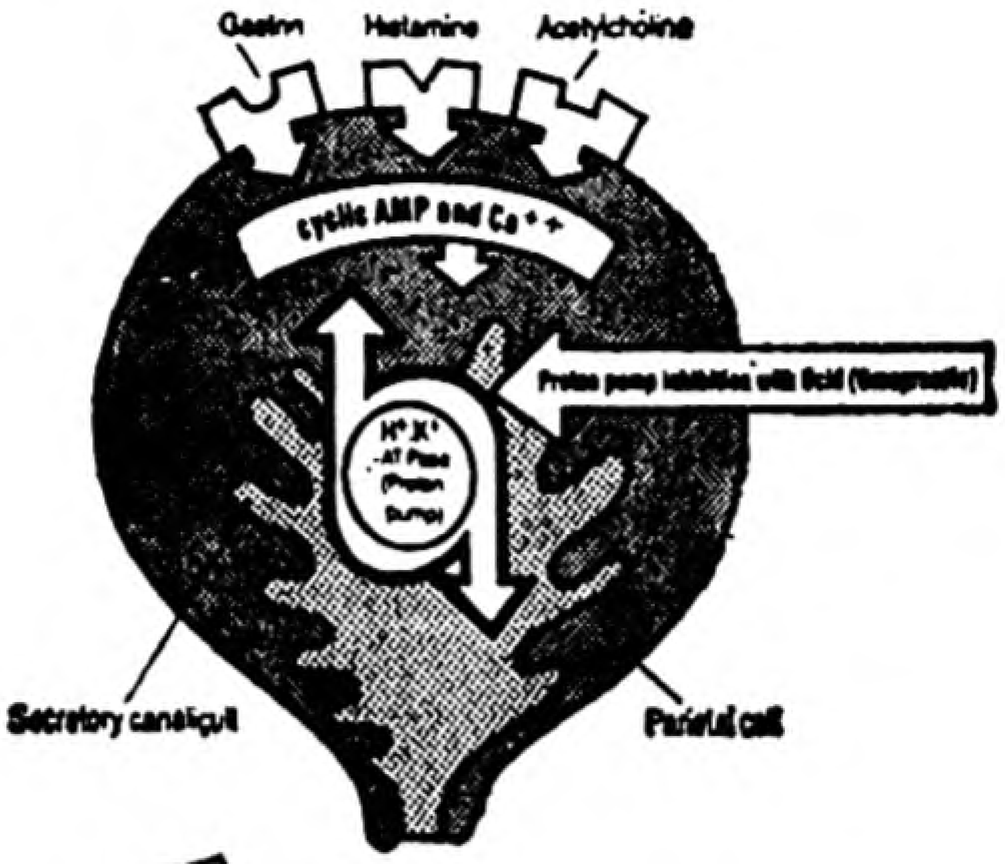
THE LATEST CONCEPT IN ACID INHIBITION



Gastrin
Histamine
Acetylcholine
cyclic AMP and Ca++
Proton pump inhibition with Ocid (Omeprazole)
H+K+-AT Pase (Proton pump)
Secretory canaliculi
Parietal cell

Ocid
(Omeprazole)

A TOTALLY NEW CONCEPT IN ACID INHIBITION

Unlike H_2-antagonists Ocid effectively blocks the final pathway of acid secretion that is common to all the three receptors
- The proton pump

* Superior healing capacity
* Faster relief from symptoms
* Effective even in resistant ulcers
* Reduced antacid consumption

In peptic ulcers, reflux oesophagitis & Zollinger-Ellison syndrome.

Ocid - The new generation antiulcerant.

Cadila

thyroiditis, pernicious anemia, Addison's disease, Diabetes mellitus and frequently internal malignancy and malignant melanoma [12,22-25].

Differential diagnosis

Vitiligo is to be differentiated from:

- Pityriasis alba
- Pityriasis versicolor
- Hypopigmented macules of leprosy
- Postinflammatory leukoderma.

Treatment

Treatment of vitiligo is not satisfactory and patients mostly are best advised to seek cosmetic camouflage for the lesions on exposed areas of body. However vitiligo is a significant cosmetic problem in people with dark complexion in whom disease causes considerable emotional and social problem. In such cases attempts to achieve repigmentation of affected skin may be worthwhile. Repigmentation is caused by activation and migration of melanocytes from a melanocytic reservoir located in the hair follicles. Therefore skin with no or little hair or with white hair responds poorly to treatment. Various treatment modalities used in vitiligo are:

1. Psoralen compounds

Psoralen compounds radically increase the erythema response of skin to long wave ultraviolet light (UVA) after topical or systemic administration. 4,5,8-trimethylpsoralen and 8-methoxypsoralen combined with exposure to sunlight (PUVA Sol) or to light source providing high intensity long wave ultraviolet light (PUVA) is effective in a proportion of cases [26]. The psoralens are given in the dose of 0.6 mg/kg body weight orally, 2 hours before exposure to sunlight [26]. For limited areas of vitiligo psoralens may be used topically, however, use of topical application of psoralens is hazardous and may lead to untoward blistering of the skin [26]/

2. Topical steroids

Potent topical steroid preparations like 0.1% betamethasone valerate [27], and 0.05% clobetasol propionate [28] are effective in producing repigmentation of areas of vitiligo. The use of such potent topical steroids, however, are known for producing local atrophy and telangiectasias. Significant systemic absorption may lead to suppression of the pituitary adrenal axis.

Systemic steroids are reported to have no place in the treatment of vitiligo [29]. However a new treatment modality "oral mini-pulse therapy with betamethasone" has recently been reported to be effective in patients having extensive or fast spreading disease [30]. In this form of treatment patients are given 5 mg of betamethasone/dexamethasone as a single dose after breakfast on 2 consecutive days per week [30].

3. Grafting and transplantation:

Several surgical procedures have been developed for treating refractory vitiligo which include grafting suction blistered epidermis, minigrafts, and transplantation of in-vitro-cultured epidermis bearing melanocytes [31-35].

4. Human placental extract

Melagenin, a Cuban medicine prepared from human placental extract and reported to contain a lipoprotein fraction, has been claimed to stimulate melanin synthesis, melanocyte proliferation and repigmentation of vitiligo macules. Though it undoubtedly is a most ineffective way to procure repigmentation in vitiligo patients, anecdotal claims are used to promote placental extract in underdeveloped areas like far East, India, Pakistan, South American and African countries as guaranteed medicine for stimulation of pigmentation [34].

5.Bleaching

For those patients having widespread disease with only few residual areas of relative hyperpigmentation, bleaching with 20% monobenzyl etherof hydroqunon may be used to achieve permanent depigmentation of these hyperpigmented areas [37]

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Original Article

Clinical Spectrum of Sick Sinus Syndrome - With Follow up

Gazanfar Ali, M A Kamili, Samia Rashid, G.Q. Allaqaband

Abstract : The clinical spectrum of the sick sinus syndrome (SSS) is described in a series of 41 patients with persistent sinus bradycardia (n=14), sinus arrests and sinoatrial blocks (n=24) and sinus bradycardia with episodic supraventricular tachycardia (n=3). Permanent pacemakers were implanted in 31 cases and are being followed up. Ischaemic heart disease and hypertension were the commonest association, with symptoms of bradycardia and tachycardia. Post-pacing the patients were asymptomatic, but 3 required antiarrythmics for tachyarrythmias. Two patients died within the follow-up period of 38 months. The ten patients who were not paced were followed for a minimum period of 24 months on medical therapy. **JK Practitioner** 1995; 2:101-3.

Key words : Bradycardia-tachycardiasyndrome, Sinus arrest, Sinoatrial block, Pacemakers.

Introduction

The terms "Sick Sinus Syndrome" [1-3] "Sluggish Sinus Syndrome" [4] and "Sino-atrial Syndrome" [5] have all been used to describe unexplained persistent sinus bradycardia with occasional episodes of supraventricular tachyarrythmias. When complicating tachyarrythmias are present the condition is called "Bradycardia-tachycardia Syndrome" [6].

We report the clinical spectrum of 41 patients with sick sinus syndrome (SSS), and also the symptomatology, associated cardiovascular abnormalities, and follow-up of paced as well as non-paced patients.

Patients and Methods

Patients treated and followed up in Medical College Hospital Kashmir (India) for SSS were reviewed. They were divided into three groups on basis of electrocardiographic manifestations into 3 groups. Group I included 14 patients with persistent and unexplained sinus bradycardia, with the heart rate varying from 40-50 beats/min. Group II included 24 patients with episodes of sinus arrest or sino-atrial blocks with A-V junctional or ventricular escape beats. Group III, included 3 patients with bradycardia-tachycardia syndrome (BTS). Each patient had at least one documented episode of either paroxysmal atrial tachycardia (n=2) or atrial fibrillation (n=1); in addition to severe sinus bradycardia.

Results

The series included 33 men and 8 women with the age range of 41-70 years (mean 56 years). Table-I shows the sex, age, age of onset of either symptoms or electrocardiographic diagnosis of the syndrome.

Data	Group		
	I	II	III
Males (No.)	12	20	01
Mean age (yrs)	59	61	68
Range	50-70	41-70	58-60
Age at onset (yrs)	56	56	56
Follow-up (yrs)	02	02	02

Table-1. Characteristics of 41 patients with sick sinus syndrome.

The diagnosis of coronary artery disease was made in 13 patients on the basis of either historical or electrocardiographic evidence. Other associated diseases included hypertension (n=8), diabetes (n=8) and chronic obstructive airway disease (n=4).

Three patients were symptomatic prior to the diagnosis for 1-26 years (mean 28 months). SA attack was the most frequent complaint in 23 patients (56%) whereas giddiness and syncope in 15 patients (36.7%) were next in frequency. Palpitations were the main complaint in 20 patients (48%) with one patient presenting with congestive cardiac failure. Associated electrocardiographic abnormalities observed in 8 patients are depicted in table-2.

Of the 41 patients, pacemaker was implanted in 31 patients to prevent SA attacks, syncope and dizziness. 3 patients with BTS were also put on antiarrythmic drugs after pacing. 10 patients were given only pharmacotherapy for their symptoms. Of the 10 patients, 6 patients remained asymptomatic on orciprenaline and in 4 patients attempts to increase

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heart rate by sympathomimetic amines were without benefit. All these 10 patients could not be paced as they could not afford to meet the cost of pacemakers.

The patients who underwent pacing remained asymptomatic, though one patient from group III, continued to have arrythmias even on adequate doses of antiarrythmic drugs and required hospital admission twice to control the tachyarrythmias.

There were also two deaths in the follow-up period in paced group, one each due to carcinoma of head of pancreas and anterior wall Q wave myocardial infarction while 3 deaths occurred in the non paced group.

Abnormality	Number
Conduction disturbance	08
AV blocks:	
First degree AV block	02
With left axis deviation	01
Bundle branch blocks:	
Right bundle branch block	03
With left ant. hemiblock	02
Left bundle branch block	01
Intaventricular conduction defect	01
Left axis deviation	10
Left ventricular hypertrophy	02
Myocardial infarction (old)	03

Table 2. Associated ECG abnormalities in 41 cases of sick - sinus syndrome.

Discussion

Sinoatrial node, though described in 1906 [7] and with its altered function having been recognized long back [8-10] with Laslett in 1909 reporting symptomatic sinus bradycardia with SA attacks, it was only in 1916 that Levine [11] first described the electrocardiographic aspects of SA exit block. A symposium on SSS was described in 1973 by Ferrer [12]. BTS was first described by Short in 1954 [13].

The clinical spectrum of SSS seen in this series is wide, ranging from sinus bradycardia without symptoms to recurrent attacks of syncope, SA attacks and congestive failure comparing well with series reported earlier [14]. Six patients of asymptomatic sinus bradycardia were included as SSS [3,14].

The recent descriptions [15,16] of SSS emphasize its frequent association with unconsciousness and need for long-term pacing [7]. Six of our patients were asymptomatic while the symptomatic group had disturbances of consciousness of variable severity (56% SA attacks and 36% syncope and giddiness). The diagnosis of CAD in our series was made in 21 (50%) cases only, with no specific diagnosis in 20 patients.

Multiprogrammable VVI pacemakers were implanted in 31 patients. It effectively controlled the symptoms of bradyarrythmias and patients with BTS were also relieved of their symptoms though in conjunction with antiarrythmic drugs. Atrial pacing for patients with SSS has also been suggested [17]. More sophisticated physiological pacemakers though effective in SSS, were not used by us because of high price. Our study reveals that VVI pacemaker is still an economical and satisfactory mode of treatment in SSS without any adverse effects in the follow-up. This is also previously reported in may Indian series [16,18].

The survival rate was good (95%) after maximum follow-up of 38 months. This contrasts with series of Wohl et al [19] who reported only 58% survival at the end of 25 months follow-up. We did not encounter any pacemaker complication. The number of SSS paced patients is increasing and it encompasses as many as 50% of pacemaker population [20].

We conclude that asymptomatic SSS detection may increase the number of SSS population. VVI mode of pacing is satisfactory, economical and improves quality of life in symptomatic patients and affects new hopes by increasing survival.

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Original Article

Clinicopathological Spectrum of Nephrotic Syndrome in Kashmir

*Parvaiz Ahmad Koul, G. H. Malik, A. G. S. Pathania**

Abstract : 102 consecutive patients (79 adults & 23 children; median age 26.5 yrs) with nephrotic syndrome were studied. Males were affected about twice as females. Majority presented with clinical features of fluid retention. Hypertension and hematuria were seen in 52.9% and 66.6% cases. Infections were seen in 18.6% cases, and hypercholesterolemia in 46.1%. Renal impairment was seen in 39.2% cases. Histologically MCGN was the commonest subtype (25.3% adults & 60.8% children). Other histological types seen in adults were DPGN (19.7%), FSGS (14%), Memb GN (13%), MSGN (7.6%), lupus nephritis (3.7%), CGN (2.5%), amyloidosis (2.5%), Cr.GN (1.3%) and focal glomerulonephritis (1.3%), whereas in children the other histological types were Memb GN (13%), Cr GN (8.7%), MSGN (8.7%), focal prolif GN (4.4%) and DPGN (4.4%). JK Practitioner 1995; 2:104-8.

Key Words: Nephrotic syndrome, Glomerulonephritis, Renal failure.

Abbreviations: NS-Nephrotic syndrome, GN-Glomerulonephritis, MCGN-Minimal change glomerulonephritis, FSGS-Focal segmental glomerulosclerosis, DPGN-Diffuse proliferative GN, MSGN-Mesangioproliferative GN, CGN-Chronic GN, Cr GN-Crescentic GN, Memb GN - Membranous GN, MPGN - Membranoproliferative GN.

Introducxtion

Nephrotic syndrome (NS) is a clinical entity characterized by proteinuria, hypoproteinemia, hyperlipidema and edema, the hall mark being heavy proteinuria in excess of 3.5 g per 1.73 m^2 surface area per day [or a protein concentration rate of >0.4 mg/mmol creatinine] [1]. All the subsequent clinical features can be attributed to the primary abnormality of protein excretion. The usual complications of the disorder [2] are:

a. Hypovolemia leading to postural hypotension, circulatory collapse and renal failure.
b. Diminished resistance to infection leading to primary peritonitis, cellulitis, septicemia and urinary infection.
c. Protein depletion leading to osteoporosis, cutaneous striae and muscle wasting.
d. Altered blood coagulability leading to arterial and venous thrombosis.
e. Reduced renal function leading to uremia & all its consequences, hypertension and all its consequences.
f. Miscellaneous - in the form of secondary renal tubular disorders, hypothyroidism, hypoferremia and hypozincemia.

NS can result from a variety of primary and secondary glomerulopathies and the histological spectrum varies from one place to the other. No study on the spectrum of NS is available from our State and the present study is an attempt to unravel the etiopathological spectrum of this clinical entity in the valley of Kashmir.

Material and Methods

The patients included 102 adequately biopsied patients of NS admitted to the renal unit of SKIMS. NS was defined as a combination of:

a. A 24 hr. Urinary protein excretion of 50mg/kg body weight or more/or 3.5 gm per 1.73 m^2 surface area.
b. Serum protein levels of < 6.0 g/dl.
c. Serum albumin levels of < 3.0 g/dl.

After a detailed clinical assessment, various investigations performed included: Urine Analysis (Aution analyzer) and direct microscopy of spun sediment, Urinary proteins & creatinine, complete hemogram (Coulter counter), serum , glucose, albumin, cholesterol (Hitachi 704 multichannel analyzer), radiographs of chest/abdomen, and ultrasonography of the abdomen for renal size or a 5 minute nephrogram. Immunological studies included assessment of C3-complement, ANA, AMA, SMA, PCA. A coagulogram was obtained and a tru-cut renal biopsy was performed under local anaesthesia. The specimen obtained was examined under a dissecting microscope for presence of glomeruli. The specimen was divided

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into 2 parts - one part of the specimen was fixed in 10% formation and processed for routine histology (sections stained by H & E, PAS and Jones methanamine silver stains). Other investigations were performed when indicated. Values have been expressed as mean ± SD. Correlation amongst variables was tested by deducing Pearson's correlation coefficient.

Results

102 patients included in the study consisted of 79 adults (56 males) and 23 children (11 males) with age ranging from 5-66 years (median 26.5 Yrs). Majority of the patients presented with facial puffiness and pedal edema. Other clinical features encountered are depicted in table 1.

Clinical Features	No (%)
a) Fluid retention	
Pedal edema	100 (98)
Periorbital puffiness	84 (82.3)
Ascites	35 (34.3)
Pleural effusions	16 (15.6)
Congestive heart failure	20 (19.6)
b) Oliguria	88 (86.2)
c) Hypertension	54 (52.9)
d) Infections	19 (18.6)
e) Hematuria	68 (66.6)
f) Thromboembolic episodes	
Renal vein thrombosis	04 (3.9)
Middle cerebral artery thrombosis	03 (2.9)
Myocardial infarction	02 (1.9)
Pulmonary embolism	01 (1.9)
g) Others	
Hepatomegaly	23 (22.5)
Anemia	12 (11.7)
Splenomegaly	05 (4.9)
Lymphadenopathy	05 (4.9)
Purpura	05 (4.9)
Arthritis	03 (2.9)
Malar rash	02 (1.9)
Chronic diarrhea	01 (0.9)
Thrombophlebitis	01 (0.9)
Bone pains & Dorsal 3rd vertebral fracture	01 (0.9)
Congenital malformations.	01 (0.9)

Table 1: Clinical features in 102 patients of Nephrotic syndrome.

Pleural effusions were related to hypoproteinemia in 14 cases and was parapneumonic and tubercular in one case each. Hematuria was mostly microscopic but was gross in 8 cases. Generalized lymphadenopathy in the 5 patients could be attributed to Henoch Schonlein purpura (n=3), SLE (n=1) and multiple myeloma (n=1). Arthritis was seen in 2 cases of HSP and 1 case with SLE.

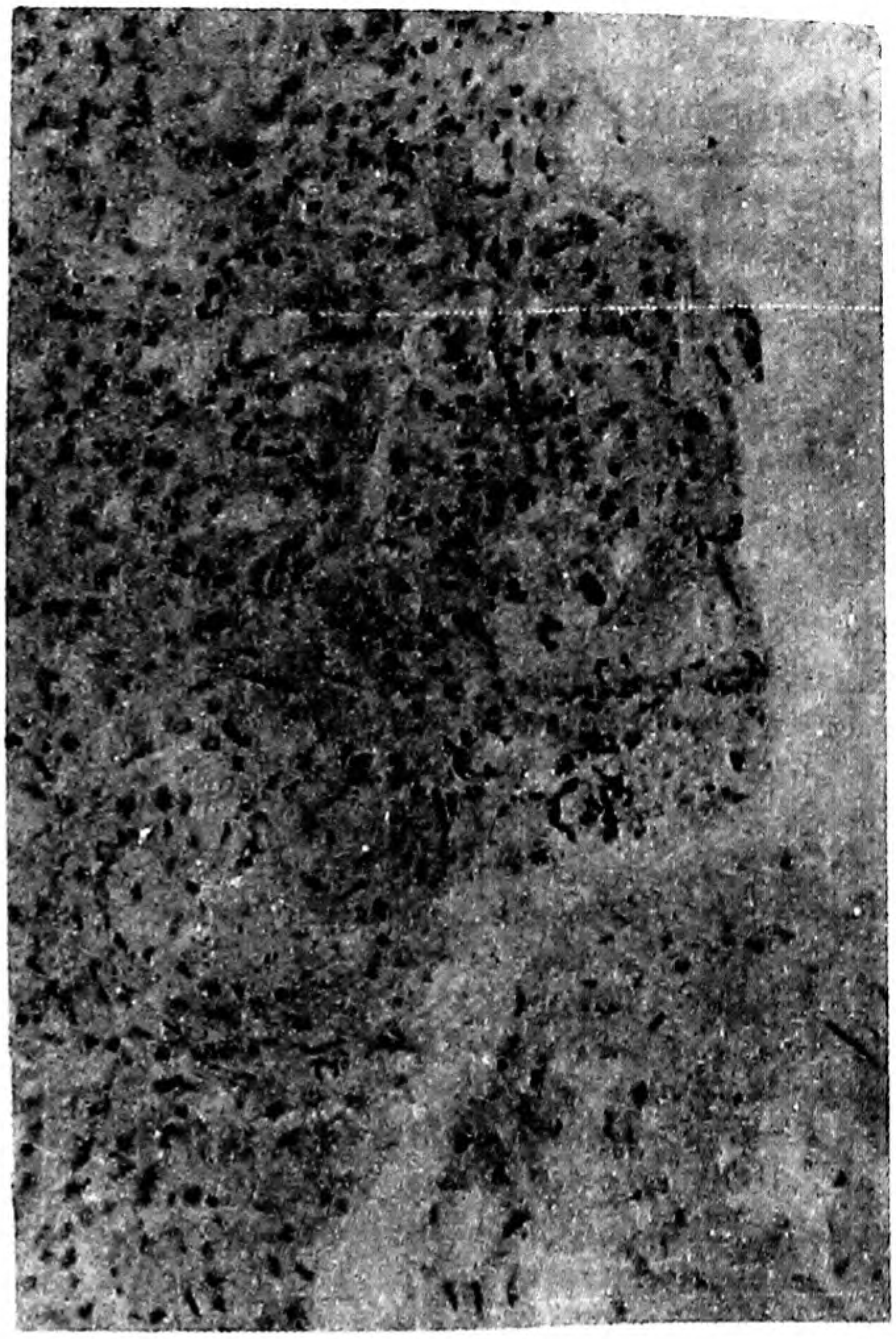

Fig. 1. Photo-micrograph showing Focal segmental sclerosis. One glomerulus has near total sclerosis and the other has only segmental sclerosis. (H&E x 100)

Splenomegaly encountered in 5 patients was attributable to amyloidosis (n=2), chronic lymphocytic leukemia (n=2) and SLE in one patient, but remained unexplained in one case. Renal vein thrombosis was suspected a clinical grounds (flank pain, gross hematuria, massive proteinuria, increase in renal size), and was subsequently confirmed angiographically in 2 cases. Bone pains and D3 vertebral fracture were seen in the patient with multiple myeloma. Congenital malformations seen were bat ears, kyphoscoliosis,subungual fibromas and sensorineural deafness in one patient. Various infections consisted of pneumonias (n=9), tubercular pleural effusions (n=1), spontaneous bacterial peritonitis (n=8), *Klebsiella* UTI (n=2) and cholangitis with *E.Coli* septicemia, and *Salmonella paratyphi* septicemia in one case each.

S.NO:	Histological Type	Total Patients Children N (%)	 Adult N (%)	 Total N (%)
01.	Total Patients	23 (100)	79 (100)	102 (100)
02.	MCGN	14 (60.8)	20(25.3)	34 (33.3)
03.	FSGS	-	11 (14.0)	11 (10.9)
04.	Memb. GN	03 (13.0)	10 (12.7)	13 (12.74)
05.	MSGN	02 (08.7)	09 (11.4)	11 (10.9)
06.	MPGN	-	06 (07.6)	06 (05.9)
07.	Focal proli.GN	01 (04.4)	14 (17.7)	15 (14.7)
08.	Crescentic GN	02 (08.7)	01 (01.3)	02 (2.94)
09.	Chronic GN	-	02 (02.5)	02 (1.96)
10.	Lupus nephritis	-	03 (03.7)	03 (2.94)
11.	Amyloidosis	-	02 (02.5)	02 (1.96)

Table-2: Histological spectrum of nephrotic syndrome.

N		Histological type
01. Systemic lupus erythematosus	3	Lupus nephritis type IV Lupus nephritis type II
03. Henoch schonlein purpura	3	Crescentic glomerulonephritis-2 Focal Prolif.GN - 1
03. Pry. Amyloidosis	2	Amyloidosis
04. Multiple myeloma	1	MCGN
05. Chronic lymphatic leukemia	1	Membranous GN
06. Behcet's syndrome	1	MCGN

Table 3. showing the underlying systemic disease with histological pattern seen on renal biopsy microscopy.

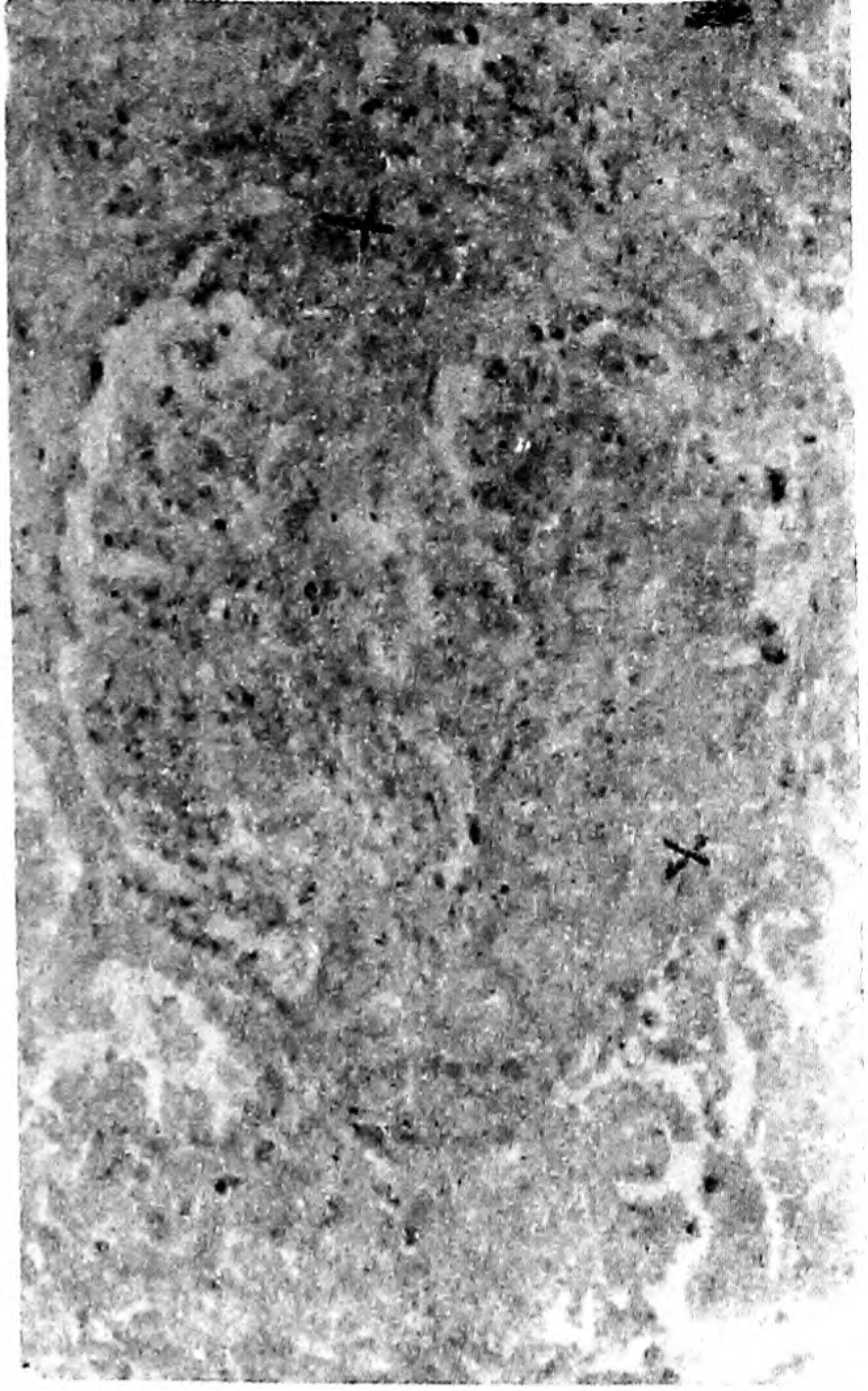

Fig. 2 Photo-micrograph showing diffuse exudative proliferative glomerulonephritis showing a hypercellular glomerulus with capsular adhesions (x). H&E x 200

Histologically MCGN was the most frequent histological type of glomerulonephritis (table 2). Of the 102 cases 11 had evidence of an underlying systemic illness (table 3) whereas the rest of the patients had a primary nephrotic syndrome. Hypertension was seen in high frequency in patients of MPGN (100%), FSGS (64%), Mem GN (61.5%), DPGN (80%), CGN (100%) and Cr. GN (66%), glomerulonephritis, but was seen in only 29% cases of MCGN. Hematuria too occurred frequently in these histological types being observed in 83%, 72.8%, 92.8%, 66.6%, 66.6%, 100% and 66.6% of cases respectively. It was found universally in cases of proliferative GN. Renal vein thrombosis complicated 2 patients of MCGN and 2 cases of membranous GN. One of these cases with MCGN had underlying Behcet's syndrome [3]. Proteinuria exhibited a wide range being 4.89±2.07 of per 24 hours in adults and 2.54±1.06 g/24 hours in children. Serum albumin levels in adults were 2.47±0.33 g/dl (range 1-3 g) and in children 2.15±0.5 g/dl (0.3-3.0 g) and did not bear any correlation with the underlying histological type. Impairment in renal function was observed in 7 children and 33 adults, being observed frequently in patients with proliferative and chronic types of glomerulonephritis compared to the rest.

Hypercholesterolemia (S. cholesterol >250 mg/dl) was observed in 14 (60.9%) children and 34 (43%) adults. Serum cholesterol levels bore a significant inverse correlation with serum albumin levels (r=-0.6126). Hypo C3 complementemia was observed in patients with lupus and membranoproliferative 6N. It was also observed in 4 cases of DPGN, and one case each of MSGN, FSGS, Membranous GN and MCGN.

In the 2 cases of lupus nephritis, the histological picture was consistent with diffuse proliferative lupus nephritis (WHO-Class IV) and in one patient with mesangial proliferative lupus nephritis (WHO-Class II).

Discussion

General clinical features of our patients resembled

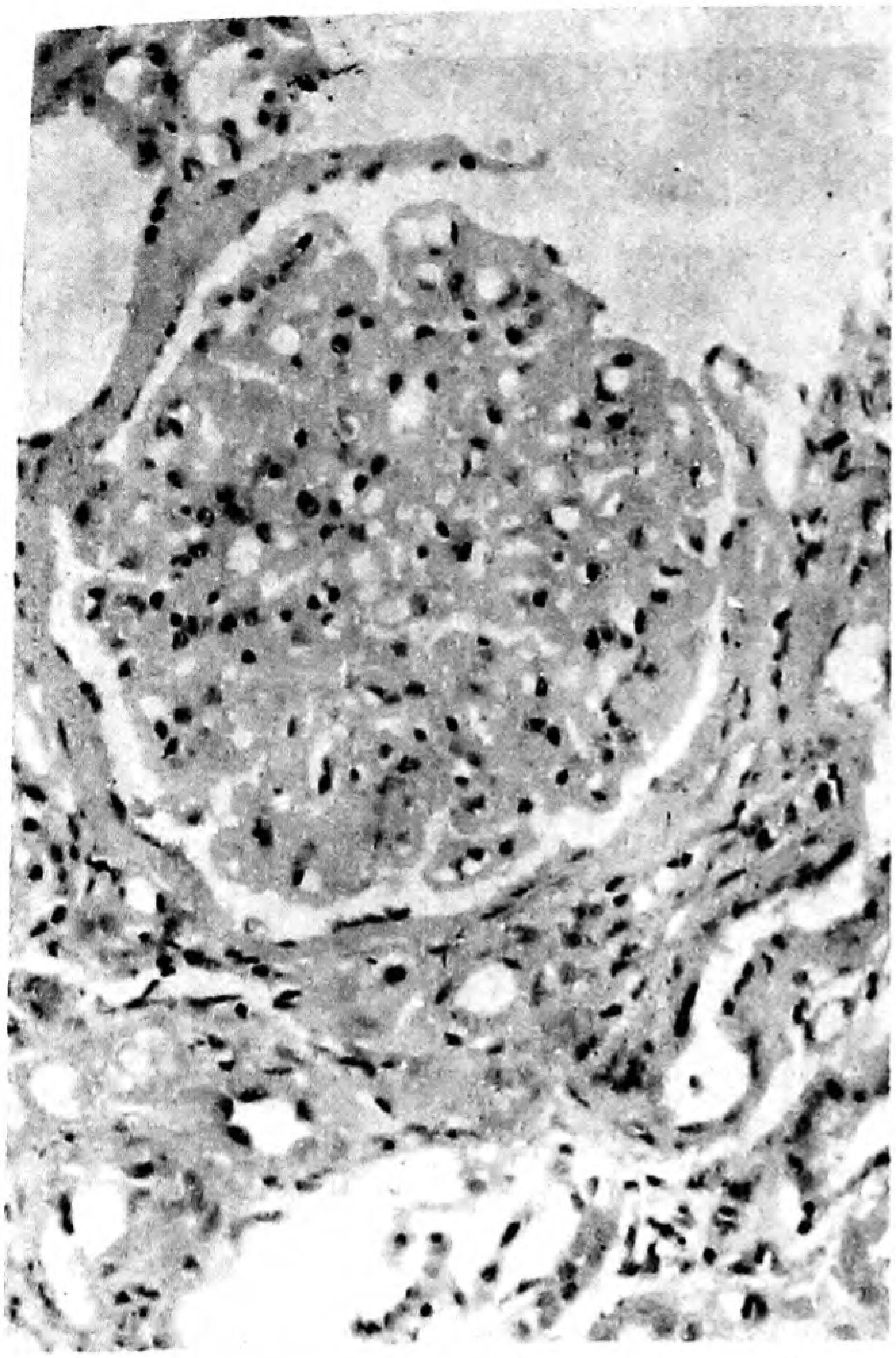

Fig. 3 Photo-micrograph showing uniform membranous thickening in a case of membranous glomerulonephritis H&E x 200

those reported earlier except in that hematuria was encountered in 66.7% cases being gross in 7.84% cases. Hematuria in our study children (65.2%) was higher than reported elsewhere. The higher prevalence of hematuria in children compared to a prevalence of 25.38-37% in other series [4-7] may be partly related to the fact that in the present series, only 60.8% were belonging to MCGN compared to more than 76% in other series [13,35]. However, even in MCGN the prevalence. of hematuria was higher (42.8% children and 40% adults).

Renal vein thrombosis (RVT) was seen in 4 of our cases, compared to a frequency of 25% reported by Chugh et al [8]. The lower prevalence of RVT in our patients can be explained due to the fact that routine angiography, as used by Chugh et al, were not used in the present study. Thromboembolic phenomena have been related to the presence of a hypercoagulable state in patients with nephrotic syndrome [9,10] One of the cases had Behcet's syndrome with MCGN [3].

The histological types of GN as compared with other important studies is depicted in tables 4 and 5. MCGN was the commonest histological type in adults as well as children, and the postulation by Johny [2] that mesangioproliferative is the commonest histological type in Indian populationcould not substantiated in the present study, though the samplesize is small to deduce any definite conclusions and would need ratification by cumulative analysis of a larger group of patients. No patient of diabetic nephropathy was observed in the present study because diabetics follow endo clinics and land up with the nephrologist only when manifest azotemia occurs, at which time a renal biopsy is rarely contemplated. The histological features resembled those reported by earlier investigations except one case had an unexplained calcification of one of the glomeruli .

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Diagnosis	Kark et al (11)	Blainey et al (12)	Sarin & Sarin (13)	Mathur et al (14)	Forland & Spargo (15)	Cameron et al (16)	Hayslett et al (17)	Shanbhag et al (18)	Johny et al (3)	Usha (20)	Present Study
MCGN	11	6	-	3	25	31	11	4	97	7	20
FSGS	-	-	-	-	-	-	-	-	54	18	11
Membranous G.N.	28	6	2	-	19	36	28	32	40	99	10
MSGN.	-	-	-	-	-	-	20	-	78	-	9
MPGN	12	-	-	-	-	12	9	6	40	20	6
DPGN.	6	6	6	-	-	47	11	13	50	2	14
Focal Prolif G.N.	-	-	-	-	1	-	-	-	35	7	1
Crescentic G.N.	-	-	-	-	-	-	-	-	5	-	1
Chronic G.N.	-	3	1	9	26	-	16	-	37	-	2
Lupus Nephritis	18	1	-	1	14	12	-	-	-	-	3
Amyloidosis	3	3	33	18	8	10	-	-	13	-	2
Diabetes mellitus	15	-	2	5	8	12	-	-	25	-	-
Others	5	4	4	17	3	9	-	5	11	5	-
Total	98	29	50	53	104	169	95	67	485	68	79

Table 4. Histological spectrum of Nephrotic Syndrome in adults

	Blainey et al (12)	Cameron et al (16)	Habib & Kleinknecht (19)	Johny (3)	Usha et al (20)	Present Study
Minimal Change GN	6	107	209	45	21	14
Focal Glomerulosclerosis	-	-	47	1	2	-
Memb. Glomerulonephritis	6	3	37	11	1	3
MSGN	-	14	21	42	1	2
MPGN	-	18	53	4	3	-
Cr. GN	-	-	-	2	2	2
Focal Prolif. GN	-	-	-	8	-	1
DPGN	6	-	-	7	3	1
Chronic GN	-	-	-	-	-	-
Others	7	39	39*	-	1	-
Total	**25**	**181**	**406**	**120**	**32**	**23**

** 20 had combined endo & extracapillary proliferative glomerulonephritis, 6 had microcystic nephrotic syndrome and 13 had unclassified lesion.*

Table-5 : Histological diagnosis of Nephrotic Syndrome in Children

causes. Amm Intern Med 1958; 49: 751-59.

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Original Article

Pattern of Gastrointestinal Tumors in Kashmir

Azra Shah

Abstract: Total of 2160 malignant tumors (65.25 percent) of the gastrointestinal tract were diagnosed out of 12969 biopsies and 3310 malignant tumors received in the department for a four and a half year period. Esophageal cancers accounted for 50.27 percent, gastric cancers 40.70 percent and colorectal cancer 8.75 percent. The prevalence of 65.25 percent of gastrointestinal tumors and 40.70 percent of gastric tumors is markedly higher than that reported from other parts of India. The peak frequency was found to be between 41-50 years of age and the tumors were more common in males than in females. Detailed studies of dietary habits and environmental factors is essential to assess their role in causing gastrointestinal malignancies. JK Practitioner 1995; 2:109-11

Key words: Oesophageal cancer, gastric cancer, small intestinal cancer, colorectal cancer.

Introduction

The gastrointestinal tract is a common site for the development of tumors in most countries of the world. Cancer of the stomach occurs in large proportions in Japan,[1] Poland and Norway [2]. Esophageal cancer is common in Iran, Central Asia, North China, Eastern and Southern Africa, France and Argentina [3]. The highest rates for colon and rectal cancer are in the United States of America and the United Kingdom [4]. In Kashmir, a very high prevalence of gastrointestinal tract (GIT) cancer, especially cancer of the upper has been noted. This paper analyses the prevalence of GIT cancer from this place for a four and a half years period and compares the results with those from other parts of India.

Material and Methods

The data for the present study was collected from the records of the department of Pathology, Institute of Medical Sciences, Soura, Srinagar. The department provides diagnostic services to the Institute and other hospitals of the state. We present the data of 12969 specimens of either surgical biopsies or resected specimens, received in the department from January, 1983 to June, 1987. The coding system described by WHO (9th revision) [5] using code number ICD 150 has been utilized for classifying the tumors of the gastrointestinal tract (Table I).

Results

Out of a total of 3310 malignant tumors during this period, 2160 were malignant tumors of the GIT consisting of 65.25 percent of all tumors. The distribution of malignant tumors of the gastrointestinal tract in the different age groups shown in Table-1.

	Oesophageal CA		Gastric CA		Small bowel CA		Colorectal CA	
ICD NO.	150		151		152		153 & 154	
Age (Yrs.)	No.	%age	No.	%age	No.	%age	No.	%age
11-20	3	(0.27)	1	(1.10)	1	(16.67)	5	(2.64)
21-30	44	(4.05)	23	(2.61)	1	(16.66)	24	(12.70)
31-40	263	(24.22)	165	(18.80)	3	(50.00)	34	(18.00)
41-50	390	(35.92)	330	(37.55)	-	-	62	(32.80)
51-60	293	(26.98)	262	(29.80)	1	(16.67)	45	(23.80)
61-70	81	(7.46;	82	(9.32)	-	-	16	(8.46)
Above 70	12	(1.10)	16	(1.82)	-	-	3	(1.60)
Total	1086	(100)	879	(100)	6	(100)	189	(100)

Table 1. showing age distribution of GIT tumors.

There were 1086 (50.27%) cases of oesophageal cancer (ICD NO. 150) (688 males) with highest frequency in the fifth decade. The youngest patient was 20 years old. Squamous cell carcinoma was the commonest histological type (79.20 percent) followed by adenocarcinoma (20.80 percent). There were 897 cases (40.70 percent) of gastric cancer, (ICD NO. 151) (687 males). The highest frequency was observed in the fifth decade. Adenocarcinoma was the commonest type (98.5 percent).

Three patients had co-existing squamous celled carcinoma oesophagus and adenocarcinoma stomach.

There were six (0.27 percent) cases of small intestine cancer (ICD NO. 152) (four males). The commonest histological type was malignant lymphoma (n

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= 5) followed by adenocarcinoma (n = 1). As per the WHO classification, peri-ampullary carcinoma was excluded from the small intestine cancer. The highest incidence of the lymphoma cases was in the fourth decade, the youngest patient being fifteen years old and oldest 55 years old. The only case of adenocarcinoma was 40 years of age.

There were 189 cases (8.75 percent) of large bowel cancer. Large intestinal tumors consisted of 70 colonic cancers (ICD NO. 153, 45 males) and 119 tumors (79 males) of the rectum, rectosigmoid and anal canal (ICD NO. 154). Of these 127 were males and 62 females. The peak incidence was in the fifth decade. The youngest patient was a 19 year old female. The commonest histological type was adenocarcinoma (93.6 percent).

Discussion

The frequency of 65.25 percent of GIT cancer is markedly high and much higher than reported from other parts of India where it ranges from 5.8 percent in Amritsar [6] to 14.36 percent in North Kerala [7].

In the present study, the relative frequency of oesophageal tumors was 50.27 percent of GIT cancer and 32.81 percent of all malignant tumors. The frequency in relation to GIT cancer is comparable to that reported from Calcutta [8] (52 percent) and Kerala [7] (54 percent) although it is lower than that reported from Bombay (72 percent) [8]. The prevalence is much higher than 31.43 percent reported from other parts of north-India [6]. The peak prevalence was in the fifth decade which is similar to that reported from Punjab [6]. The male to female ratio of 1.72:1 differs from previous Indian reports which have shown a higher male preponderance, except Prabhakar and Maingi [9] (1987) who also found an equal distribution between the two sexes.

Malignant tumors of the stomach in the present series formed 40.20 percent of all gastrointestinal tumors and 27 percent of all malignant tumors. This prevalence is higher than compared to earlier studies. Paymaster et al (1968) observed figures of 13 percent and 2 percent respectively [10] and Leena Devi and Suvarna (1980) 22.5 percent and 3.2 percent respectively. However a high frequency of 43 percent of ali GIT cancer has also been reported from Manipal [11]. A male preponderance (3.57:1) and the peak age prevalence in the fifth and sixth decades is in keeping with the age and sex patterns in other parts of the country [12]. The high incidence of stomach cancer in the present series may be due to dietary and environmental factors which have been implicated in causing gastric cancer [13].

Neoplasms of the small intestine are rare. In this study they comprised 0.27 percent of GIT malignancies, which is similar to 0.3 percent observed from Amritsar [6]. It is however much less than 3.5 percent observed by Ahuja and Chandra [14]. Malignant lymphomas constituted 83.4 percent. A higher percentage of malignant lymphomas has also been reported by other workers [14].

The relative frequency of large intestinal cancer (8.75%) observed in the present study is much lower than that observed from Bombay (20.6 percent) [15]. Two-thirds of large bowel cancer was found in the rectum and anal canal. The highest prevalence was in the 5th and 6th decades which is similar to that observed by other Indian studies.

Reports from different centers of the country have implicated smoking, betel nut chewing, alcohol, eating smoked fish, chilies and drinking hot tea [12,13] in causation of cancers. In this valley, a special type of salted tea which is boiled for hours and taken very hot may be contributing to the high incidence of esophageal cancer. However an in-depth study is required before this can definitely be implicated as a cause of oesophageal cancer. Segi et al (1957) have shown that a rice diet causes a higher risk of gastric cancer [16]. Kashmiris are rice eaters and it may contribute to the causation of gastric cancer

Our observations emphasize the need of extensive efforts to identify the high risk factors especially dietary and environmental factors of the region.

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Original Article

Clinical and Immunogenetic Profile of Rheumatoid Arthiritis Patients in Kashmir

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Abstract : Two hundred and five patients of rheumatoid arthritis, seen over a period of 4 years were studied. The disease was seen more commonly in females, and in fourth decade. The majority of the patients had relatively mild disease at the time of presentation. Small joints were mostly affected and the commonest radiological findings were soft tissue swelling and osteoporosis. Extraarticular manifestations were uncommon and vasculitis rare. Mid expiratory flow rate was the commonest lung function abnormality. 68 patients of classic and definitive rheumatoid arthritis patients were typed for four HLA-loci A, B, C and DR. Out of these 40 patients (58.82 percent) were DR4 positive test. 52 patients (76.47 percent) were rheumatoid factor (RF) positive. Of the 142 control subjects 7 cases (4.9 percent) were RF positive and 31.69 percent were DR4 positive.

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Introduction

Rheumatoid arthritis (RA) is a chronic multisystem disease. Although there are a variety of systemic manifestations, the characteristic feature of the disease is persistent inflammatory synovitis, usually involving peripheral joints in symmetric distribution. RA is the commonest of the arthritides, and exact a great toll in crippling. This comprises a group of diseases of high morbidity and low mortality, with high potential for crippling disability among a relatively younger age group. Despite advances in our understanding of the immunological processes leading to articular inflammation and connective tissue destruction in RA, the cause of this malady remains an enigma. Indeed it is still not clear whether RA is one disease with multiple etiologies or a symptom complex produced by a single causative factor. A wide clinical spectrum of RA is recognized; thus the patients selected may not represent a true cross section of disease presentation or clinical profile. In addition environmental, social, genetic and economic factors may play a role in the expression of the disease. Thus the illness may vary in severity in rather homogeneous isolated populations. Family studies indicate a genetic predisposition. The role of genetic influences in the etiology of RA was established by demonstration of an association with class II major histocompatibility gene complex antigen HLA-DR4. Severe RA is found at approximately four times the expected rate in first degree relatives of individuals with seropositive disease.

Since the etiopathogenesis of RA is unknown, geographical variations in clinical, immunological and immunogenetic pattern may provide some clues

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towards the etiopathogenesis of this disease. The present study describes these features in patients with RA from Kashmir.

Material and Methods

Over a 4 year period (October 1987-October 1991) 205 adult patients with RA attending the out patient department of Physician Medicine & Rehabilitation, Institute of Medical Sciences were studied. The diagnosis of RA was established according to the American Rheumatism Association criteria [1]. All the patients underwent a detailed clinical evaluation wherein special stress was placed on the topography of joint involvement and extra-articular manifestations. The patients were assigned an anatomical class according to the criteria of Steinbroker [2]. Apart from routine hematological and biochemical investigations, serological studies in the form rheumatoid factor (latex agglutination) and ANA (indirect immunofluorescence), were performed. 68 patients with unequivocal deforming and erosive disease were typed for four major HLA loci A, B, C and DR. 142 control subjects were also typed for HLA, A, B, C and DR.

HLA-A, B and C were typed by two stage microlymphocyto-toxicity technique and DR typing was done by Nylon wool separation of B cells and typing method.

Results:

Clinical Observation.

The patients were in the age range of 11-55 years and the median age of onset of RA was 37.19 years with a female to male ratio of 3.5:1.

The average joint count on the first visit was 7. The joints involved and their frequency is given in Table-1. 27 (13.17%) patients presented in Stage I disease, 122 (59.5%) in stage II, 36(17.56%) in stage III and 20 (9.75%) in stage IV.

Extra-articular Manifestations:

There were 32 patients (15.60%) who showed typical rheumatoid nodule. Detailed pulmonary assessment including lung function tests were performed only in 60 representative patients. Of these 47 patients had normal skiagram while 7 showed evidence of pulmonary infiltrates, and 6 had pleural effusions. Pulmonary function studies revealed reduced vital capacity in 21 patients (35 percent), reduced mid expiratory flow rate in 39 patients (65 percent) and diminished FeV1 in 12 patients (20 percent). Cardiac involvement was sought clinically and by electrocardiogram. Seven patients had prolonged PR interval, 4 had pericarditis and 2 had aortic regurgitation. 8 patients had splenomegaly and 40 patients are found to have mild to severe anemia.

Joints	Frequency of involvement in present study. (Percent)	Frequency of involvement in Northern India. (Percent)	Frequency of involvement in White race. (Percent)
Wrist	73.17	95	82
PIP	76.09	90	63
MCP	71.70	87	87
Knee	76.09	75	50
Ankle	34.14	55	53
MTP	29.26	47	48
Shoulder	29.26	40	47
Elbow	26.82	35	21
Hip	08.29	15	Not mentioned
Cervical spine	07.80	0.8	Not mentioned

PIP = Proximal Interphalangeal Joint.
MCP = Metacarpophalangeal joint.
MTP = Metatarsophalangeal joint.

Table-1: Comparison of joint involvement in rheumatoid arthritis in patients from Kashmir, Northern India and White race.

Serology:

Using the commercial latex fixation test kit, 52 (76.47 percent) patients had a positive RF, titer ranging from 1:48 to 1:92 by the dilution technique. ANA was positive in titer of 1:8 or more in 27 patients.

Table 2 shows the results of HLA typing in 68 classical and definite RA patients and 142 control subjects.

Antigen	Patient (n=68) No	%	Control (n=142) No	%	PR	X2	P
DR1	08	(11.76)	16	(11.27)	1.05	0.008	HS
DR2	29	(42.15)	62	(43.66)	0.76	0.019	NS
DR3	09	(13.24)	21	(14.79)	0.878	0.0394	NS
DR4	40	(58.82)	45	(31.69)	3.08	14.03	001
DR5	08	(11.76)	20	(14.08)	0.81	0.2153	NS
DR6	09	(13.24)	24	(16.90)	0.75	0.4687	NS
DR7	13	(19.12)	54	(38.03)	0.385	7.57	.005
DR8	-	-	09	(06.34)	0	5.22	.025
DR9	02	(02.94)	06	(04.23)	0.686	0.46902	NS
DR10	06	(08.82)	04	(02.82)	3.34	3.34	NS
DQW1	50	(73.53)	102	(71.83)	1.089	0.066	NS
DQW2	19	(27.94)	73	(51.41)	0.366	10.28	.005
DQW3	51	(75.00)	72	(50.70)	2.926	11.18	.005

HS = Highly significant, NS = Non-significant

Table-2: HLA-DR antigen in the patients & control subjects

Discussion

There are some important differences in clinical presentation of RA patients in Kashmir and Northern India on one side and the patients of European origin on the other. First the anatomic stage at which our patients presented indicates a milder form of joint disease than that seen in the west. The present study also shows that the extent of severity of extra-articular manifestations of RA in patients from Kashmir and Northern India is less than that seen in the west. Only 32 (15.60 percent) of our patients had subcutaneous nodules in contrast to the accepted figures from of 20 to 35 percent reported from the white race. Malaviya et al [3] have reported subcutaneous nodule in 8.5 percent patients and Walker and Wright [4] have reported subcutaneous nodules in 44 percent male and in 24 percent of female patients. Systemic vasculitis was not found in any of our patients. Eye involvement in the form of episcleritis was noted in 5.8 percent of our patients. Similar results have been reported by Walker and Wright [4]. Reddy, et al [5] examined 50 patients of RA and reported 33 percent patients having one or more ocular changes. Cardiac involvement was found in 19 percent patients, 10.29 percent patients having electrocardiographic evidence of cardiac involvement in the form of PR prolongation and about 3 percent patients had aortic regurgitation. These patients did not have any history of rheumatic heart disease or any other cause of aortic regurgitation. Pericarditis was clinically diagnosed in 5.88 percent patients and was substantiated by electro and echocardiogram. Malaviya et al [3] found mitral regurgitation in two cf 258 patients studied. Lebowitz [6] reported pericarditis in 50 percent of patients at autopsy. Pulmonary involvement was seen in 6.34 percent of our cases in the form of pleural effusions (n=6), and interstitial lung disease 9n=7). Lung function tests revealed reduced vital capacity in 35 percent patients, reduced mid expiratory flow rate in 65 percent, and diminished FeV1 in 20 percent patients. Malaviya et al [3] have found reduced vital capacity, reduced mid expiratory flow rate and reduced FeV1 36 percent, 92 percent and 30 percent patients respectively. Walker and Wright [4] have reported interstitial pulmonary fibrosis in 1.6 percent patients. Splenomegaly was noticed in 8 (11.76) patients. Walker and Wright [4] have found splenomagly in 3 percent patients.

The frequency of seropositivety and the association of disease with DR4 is comparable to that reported in Caucasians. 40 (58.82 percent) of 68 patients in our study were DR4 positive compared to 31.69 percent controls ($p<.001$). 52 (76.47 percent)patients were RF positive as compared to 7 (4.9 percent) control subjects. No significant difference was found in antigen frequencies of HLA-A, B and C loci in patients and controls. The high frequency of HLA-DR4 antigen detected in a representative group of Kashmiri population correlates well with other North Indian studied by Malaviya, et al and Mehra, et al [3,7] as well as those in white caucasoid population [8-12] but is in conflict with observations of Michol and Woodrow (13,14) who reported a strong association of DR1 with rheumatoid arthritis in Indians from Liver pool.

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Orginal Article

A Study of the Present Magnitude of Psychiatric disorders and existing treatment services in Kashmir (1990-94)

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Abstract: Out of a total of 41,542 new patients treated at the hospital for Psychiatric diseases, Srinagar (Kashmir), during the past five years (1990-94), the least number 1762 (4.24%) of patients reported for the treatment during the year 1990 and the number of the patients increased nearly ten fold to 17,584 (42.33%) in the year 1994. Possible reasons for this marked increase when compared with a more or less constant rate of patients over the five year period, a decade earlier, from 1980-84; have been discussed. The adequacy of the presently existing treatment services and the comparison of work output by the clinical staff of the department of Psychiatry, GMC, Srinagar with that of the Psychiatry department at PGI, Chandigarh is presented. JK Practitioner 1995;2: 115-7.

Key words : Mental disorders, variation, treatment services, work-load, output.

Introduction

Life stress is a product of changes that occur in one's life, that require adaptations, coping and social readjustment [1]. The review of epidemiological studies of life events and psychiatric disorders has shown that 32% of the psychiatric cases (41%) among females can be attributed to stressful life events [2]. Accumulation of severe or numerous life events can act as strong stressor and affect the psychiatric equilibrium, producing maladaptive patterns of behaviour and possibly mental disorders [3].

Exposure to a variety of severe traumatic stressors has at present become almost a daily affair for the majority of population living in Kashmir, irrespective of their age, sex, domicile, profession or socioeconomic status [4]. Various newspapers and magazines have from time to time reported an alarming increase in mental problems during the previous five years of turmoil in Kashmir [4-6]. So far, however, no systematic study has been reported on the subject. Since research has an important bearing on the services rendered [7]; and careful analysis of the data pertaining to the outpatient population of a hospital or institute, as described earlier by our group [8,9], can in addition to theoretical implications of etiology, help construct a 'client profile' to keep the services strictly relevant to the needs of the community [10,11]. As such it was considered pertinent to undertake the present investigation with a view to assess the magnitude of increase in mental disorders over the period of past five years. and to review the existing treatment services in Kashmir

Material and Methods

All the patients who sought treatment at the outpatient department of the sole provincial level Psychiatric diseases hospital of Government Medical College Srinagar, Kashmir, from Ist January 1990 to 31, December 1994 formed the sample for the study. Both the categories of patients - those who after detailed evaluation and consultant's opinion got registered with OPD of the hospital, as well as those patients who received treatment on 'walk in clinic' basis on OPD tickets, without being registered, were included in the study. The relevant details about the sample were obtained from the record section and OPD ticket counter of the hospital OPD, and the data thus obtained was analyzed.

Since the outpatient department remains open six days a week (emergency services run 24 hours each day), the average number of the patients examined per day during each year was, therefore, calculated on the basis of approximately 315 working days per year 1980-84; and keeping in view the present prevailing conditions, 300 working days per year during 1990-94.

Results and Discussion

Out of 41542 total new patients (Table-1) treated during the study period, 1762 (4.24%) reported for treatment during the year 1990, and thereafter there was a steady increase in the number of patients; so much so that within a span of only 4 years the number increased nearly ten fold to 17584 (42.33%) in 1994, compared to 16079 patients treated 10 years from 1980-84 (Table-II). The number of new patients per day remained more or less constant over 1980-84, with an average of 9-10, but in 90's, the number of

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patients per day demonstrated a rise from an average of about 6 patients per day in 1990 to about 59 new patients per each working day in 1994. This steep increase in the number of patients over the past five years may be attributed to several reasons. It is a well documented phenomenon that when a population gets suddenly exposed to catastrophic stress, the people with less intense psychiatric disorders like anxiety, hysteria and mild depression tend to ignore their symptoms initially, while as the people with major mental problems like schizophrenia and mania continue to seek or are made to seek treatment. However, in case of continued stress not only do the existing patients come forward for treatment but more and more apparently normal people suffer from palpitations, insomnias, anxiety disorders, hysteria, depression and even major psychiatric disturbances and seek help [12]. Since in 1990 the whole population of Kashmir got abruptly exposed to severe stress arising out of mass destruction of life and property, overwhelming fear and uncertainty [13], the psychological response of the people with psychiatric morbidity as reflected in the OPD population appears to be in complete conformity with the aforementioned assertion. As these stresses have almost become a perpetual process over the years the psychiatric disorders are assuming fulminating dimensions.

Year	Registrations No (%)	'Walk in clinic' No (%)	Total pts. treated No (%)	Average no of new pts treated per day.
1990	234(06.29)	1528 (04.04)	1762 (04.24)	06
1991	761(20.46)	1394 (19.55)	8155 (19.63)	27
1992	616 (16.56)	3699 (09.78)	4315 (10.39)	14
1993	584 (15.70)	9142 (24.17)	9726 (23.41)	32
1994	1525 (40.99)	16059 (42.46)	17584 (42.33)	59
1990-94	3720 (100)	37822 (100)	41542 (100)	28
Reg/Not Reg Ratio 1990-94	9%	91%	100%	

Table 1 : **New patients treated during Jan 1990 - Dec 1994.**

Table 2: **New patients treated during Jan 1980 - Dec 1984.**

Table 3: **Comparison of clinical staff strength of the departments of Psychiatry, GMC Srinagar and PGI-Chandigarh.**

Year	Registrations No (%)	'Walk in clinic' No (%)	Total pts. treated No (%)	Average no of new pts treated per day.
1980	1294 (27.74)	2621 (22.96)	3915 (24.35)	12
1981	1030 (22.08)	2456 (21.52)	3486 (21.68)	11
1982	906 (19.43)	1662 (14.56)	2568 (15.97)	08
1983	764 (16.38)	2139 (18.74)	2903 (18.05)	09
1984	670 (14.77)	2537 (22.22)	3207 (19.95)	10
1980-84	4664 (100)	11415 (100)	16079 (100)	10
Reg/Not Reg Ratio 1980-84	29%	71%	100%	

Table 2:

	GMC - Srinagar			PGI Chandigarh*
Posts	19080-84	1990-94	1995	1995
Professor	One	One	Vacant	One
Additional Professor	nil	nil	nil	two
Associate Professor	one	one	vacant	two
Assistant Professor	one	nil	nil	three
Lecturer	nil	two	two	nil
In Clinical Psychology	one lecturer	vacant	vacant	three additional Professors & one Assistant Professor.
Senior Residents	two	two	vacant	nine
Junior Residents	two	two	two	eleven
Assistant Surgeons**	two	two	two	nil

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* Souvenir-cum-abstracts - International symposium on Cultural Psychiatry 1995, Chandigarh.

** Also engaged in assisting the hospital administration in the management of stores, diet, vehicle etc.

Table 3:

Inspite of a ten fold increase recorded in the number of the patients during a short span of 4 years, it is possible that this number represents only a fraction of the actual number of patients seeking treatment for mental and emotional disorders in Kashmir. Undoubtedly, even under normal circumstances, most of the psychiatric morbidity especially in rural or lesser psychologically sophisticated population is often missed or misdiagnosed [15]. Furthermore, the symptoms of mental disorders quite often express themselves through somatic body language in the form of somatization, psychosomatic complaints, hypochondria and multiple physical symptoms; such patients wander from one medical speciality to other, undergo a lot of physical diagnosis, sometimes they even undergo a lot of physical investigation, at times even surgery without satisfactory results. However, as is evident from a recent study of the authors and associates [9], a better psychiatric awareness and increasing acceptance of this facility by the community is bound to unmask the real magnitude of these emotional disorders in Kashmir. The social metamorphosis due to present prevailing conditions is also likely to contribute to it [16].

Department/ Services	Department of Psychiatry GMC-Srinagar	Department of Psychiatry PGI-Chandigarh
OPD DEPARTMENT	Open 6 days a week	open 6 days a week
Detailed evaluation	1525	1800
Walk in clinic (1994)	16059	5000
Follow up Visits	23000	20000
IN-PATIENTS		
Bed strength	100	40
TEACHING AND TRAINING		
Undergraduates	Yes	Nil
Postgraduates	Yes	Yes

Table 4: **Comparison of teaching and treatment services available through the Department of Psychiatry of GMC-Srinagar and PGI-Chandigarh (1994)**

Another striking finding of our study (Tables-3 & 4) is that the treatment services are quite inadequate and the available clinical staff is far less than is normally expected or recommended. No mental health, services whatsoever, are available at the periphery at primary health centre or district hospital level. Ironically the staff strength of the sole psychiatric services through the department of Psychiatry of GMC, Srinagar, catering to the needs of whole Kashmir valley, adjoining areas of Jammu province and Ladakh, has miserably decreased with the increase in the number of patients seeking treatment. This inadequacy can be realized from the fact that only 9% patients could be registered in 1994, despite the high number of patients; and the remaining 91% of the patients, the majority of whom would have needed a detailed evaluation and adequate psychological treatment, had to be disposed off hurriedly in walk-in clinic. This paucity of staff and consequent paucity of time in the light of ever increasing needs of the community is bound to affect the patient care and other related activities like teaching and research pursuits most adversely in the days to come.

A comparison of the work output of this understaffed departments of psychiatry with the psychiatry department of one of the most reputed institutes of southeast Asia - PGI Chandigarh is quite revealing and heartening. As far as the work load of patients care and teaching is connected, the department of GMC Srinagar is at par and even ahead of PGI Chandigarh. The department is managing 100 inpatient beds and teaching both undergraduate as well as post-graduates whereas the psychiatry department department of PGI has only 40 inpatient beds to look after and undertakes only postgraduate teaching. However, the sphere in which the department of GMC is lagging far behind the department of PGI Chandigarh is in the field of research pursuits. Some of the possible reasons for this disparity many include greater emphasis on research in PGI compared to our department where research is of only auxiliary importance. Moreover, the research pursuits at PGI have received encouragement through sponsorship by bodies like WHO, ICMR etc., and the research oriented academic atmosphere is far less developed in our setting than at PGI Chandigarh. The limitations of this presentation is that the amount of increase in the number of the emotionally disturbed patients seeking treatment psychological problems at other associated specialities and private consultation services is not incorporated in it. The authors are studying the data which will be reported elsewhere. This study is a preliminary step in assessing the magnitude of increase in mental disorders and the adequacy of existing treatment services in Kashmir. It is an area which needs to be explored both in its depth and breadth. It would aid planning future strategies for the achievement of the objectives of National Mental Health Programme (NMHP) and the Alma Ata declaration "Health for all by the year 2000 AD," and help to meet the challenge of ever increasing mental morbidity of the populations in this region. It will also help in decreasing stigma and increasing knowledge among the lay public, so that those who suffer from depression, anxiety or schizophrenia will not have to suffer from shame and guilt (17).

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Lung Cyst in a young farmer

Parvaiz Ahmad Koul, A. Wahid

(JK Practitioner 1995;2: 118-9)

A 30 year old farmer presented with cough of 2 months duration unresponsive to routine medications including antibiotics. Clinical examination of the chest revealed an impaired note, decreased VF/VR, and a diminution in the breath sounds. Rest of the physical examination was unremarkable. Hb was 12.6g/dl, TLC 6.4 X 10^9/l, DLC P60, L38, E2, and platelets 210 X 10^9/l. Serum biochemical parameters were normal. Sputum cytology, Gram's and AFB smear were negative. An ultrasound examination of the abdomen was normal. Radiograph of the chest is shown in Fig 1. Casoni's test and IHA were performed and a CT scan was done.

Diagnosis

Water-lily sign in hydatid cyst of the lung.

Posteroanterior chest film demonstrated a cystic lesion in left upper zone with a irregular fluid level. Casoni's test was positive and IHA was suggestive of hydatid disease.

The lungs are the second most frequent site of hydatid cyst in adults [1] and the most common in children [2]. Most intact cysts are asymptomatic and found incidentally on chest radiography, but occasionally symptoms occur due to pressure effects on adjacent structures [3]. Cyst rupture can result in sudden cough, hemoptysis, and expectoration of cyst fluid, membranes, and scolices [3]. Superimposed infection produces fever and purulent sputum. Eosinophilia occurs in about 40% of patients and positive serology in about 80% [3]. False negative serology occurs mostly with unruptured cysts, especially in children (4).

Cysts are multiple in 30%, bilateral in 20% and in the lower lobes in 60% of cases [3,5]. There is a predilection for the right lung and posterior segments [3,5]. Cysts can grow to over 20 cm in diameter, and larger cysts may displace the mediastinum or diaphragm [3,6]. A centrally located closed (i.e. unruptured) cyst is usually well defined and round. More peripheral cysts conform to pressure from adjacent structures, and cysts in the interlobar fissures resemble loculated effusions [3,5]. Cyst enlargement may produce pleural reaction and atelectasis, resulting in loss of definition of its contours (3,5). On fluoroscopy, it may alter shape with respiration like any other thin-walled cyst. Calcification is rare [3].

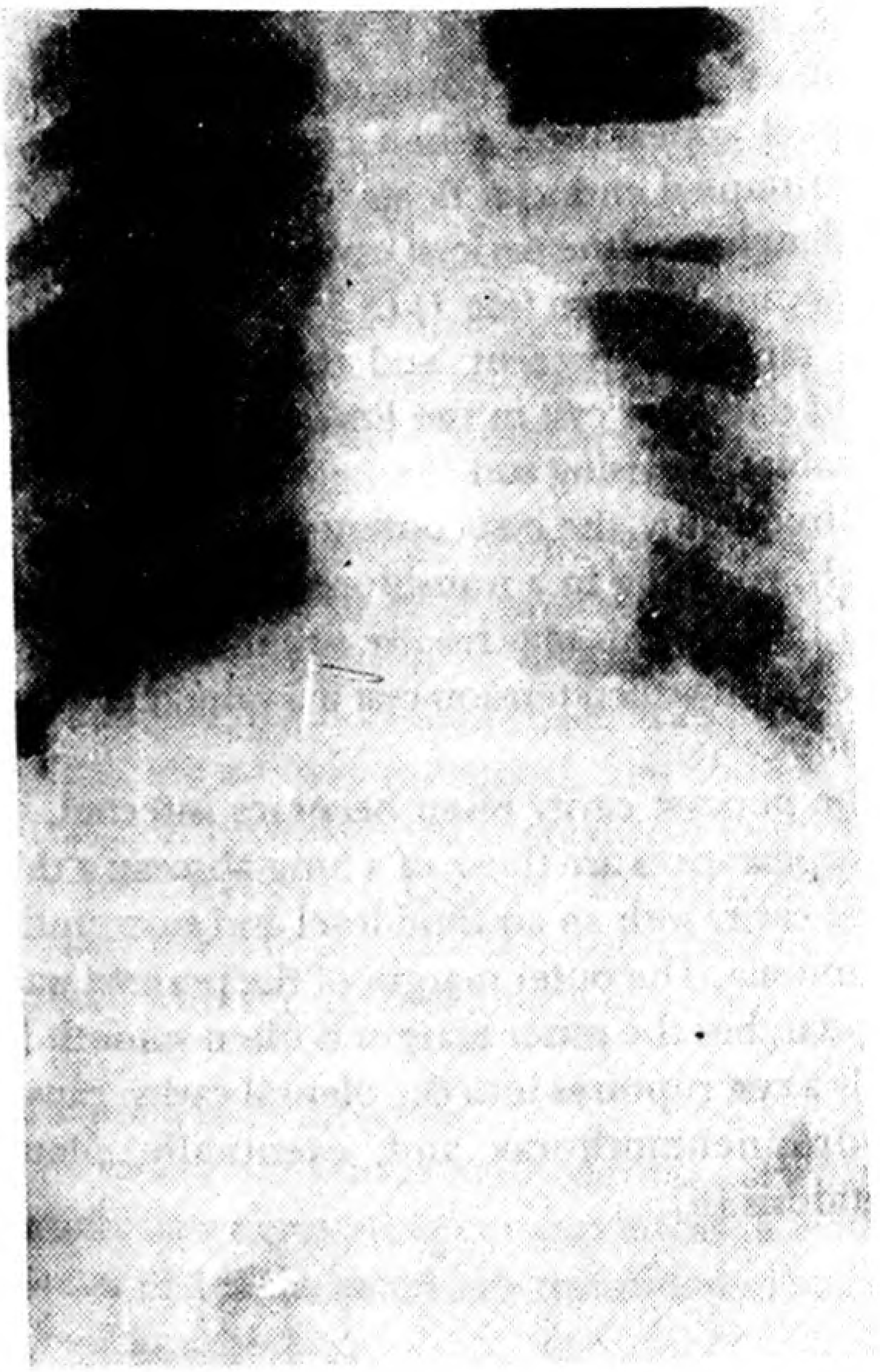

Fig. 1

Open (i.e. ruptured) cysts produce several different appearances. As the cyst enlarges, it erodes bronchioles. Air is introduced between the pericyst and laminated membrane and appears as a thin, lucent crescent in the upper part of the cyst: the crescent or meniscus sign or perivesicular pneumocyst [2,7,8]. It is a sign of impending rupture and is an indication for urgent thoracotomy[9]. As more air enters the cavity between the pericyst and the laminated membrane,

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the two separate completely and the cyst shrinks and ruptures. Cyst fluid may be expectorated or spread into the surrounding lung, provoking an allergic, eosinophilic pneumonia and even anaphylaxis. Bacterial infection may be superimposed or the cyst fluid may causes bronchopneumonia [3].

When the parasite itself ruptures and air enters it, the endocyst is outlined by a double arch of air: Cumbo's sign [3,5]. Tomography may show an onion-peel appearance. When completely collapsed, the crumpled endocyst floats freely in the most dependent part of the pericyst cavity the iceberg, waterlily, or camalote sign (Fig.1) [3,5,6,8]. Daughter cysts may, rarely, be present, and the appearance of a round daughter cyst in the lowest part of the cavity resembles the rising sun (3).

Infrequently, the cyst contents are expelled completely, resulting in a translucent cyst resembling a bulla [3,5,8]. This may resolve spontaneously or become a thin-walled tension cyst if a valve mechanism develops.

The pericyst cavity often becomes infected, and the appearances are those of a lung abscess: a thick-walled cavity with an air-fluid level and surrounding pneumonia. The outer margin of the pericyst wall is irregular, but the inner margin is often smooth [2]. Rarely a cyst ruptures into the pleural cavity, causing a hydropneumothorax and, eventually, pleural hydatidosis [8].

After a diagnosis of hydatid cyst was confirmed on CT scan and suggestive hydatid serology our patient underwent surgical management of the cyst.

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Assessment and Management of Occupational risk of Hepatitis B in Health Care Professionals

GM Malik, Parvaiz A Koul.

Extent of infection

The occupational transmission of hepatitis B was first recognized after clinical cases of hepatitis in healthcare workers were associated with outbreaks of jaundice in their patients. Subsequent studies showed that healthcare workers had a higher incidence of hepatitis B and a greater prevalence of markers of infection than those who had no occupational exposure to patients or blood products. One study in hemodialysis centers in the United States revealed that approximately half of the patients and one-third of the employees had evidence of current or previous hepatitis B infection [1]. Furthermore, the prevalence of markers indicating previous infection increased with age and/or years in occupation [2-4].

Tens of thousands of healthcare and public safety workers are infected with HBV at work every year. The exact figure is difficult to ascertain because official statistics are often inadequate and do not record subclinical infections.

It has become accepted, therefore, that hepatitis B is the major infectious occupational hazard to workers in the healthcare setting and to those who may come into contact with blood or other contaminated body fluids in their workplace.

Transmission in the workplace

HBV is present in extremely high concentrations (up to 10^{12} infectious doses per ml) in the sera of patients who are acutely infected or who are HBeAg-positive carriers. Infectious fluids include:

- blood, blood components and blood products
- semen
- cerebrospinal, vaginal, synovial, pleural pericardial, peritoneal & amniotic fluids
- other body fluids contaminated with blood.

The virus may be transmitted when contaminated fluids penetrate the skin or splash into the eye or onto the mucous membranes of a susceptible individual, by transfusion with contaminated blood, needlestick injuries and blood splashes to the eye.

Contaminated medical instruments are rarely responsible for transmission of infection in countries where equipment is sterilized and disinfected routinely. However, contaminated acupuncture needles, lancet devices for blood samples of diabetics and certain jet guns have been implicated in transmission [5-7].

HBV is not transmitted by the respiratory route, contaminated food or water, insects or other vectors. It is not present in urine or feces unless these are contaminated by blood. Saliva itself does not contain the virus, but saliva in the mouth may contain small quantities of virus excreted by crevicular fluid, bleeding gums or oral lesions. However, saliva is not thought to be important in hepatitis B transmission except in dentistry, where gross bleeding is common.

Consequences of infection

The individual worker who contracts HBV infection may face devastating consequences in terms of personal ill health, secondary transmission and possible work restriction.

Many cases of HBV infection are subclinical, but those with the full clinical manifestations can cause significant illness and, in a small percentage of cases, death from fulminant hepatitis. Illness and absence from work may be prolonged. For example, work absences of 4 to 12 months are not uncommon. Chronic infection may develop, with the consequences of cirrhosis or primary liver cancer.

Transmission of HBV from healthcare workers to patients occurs much less frequently than transmission from patients to healthcare workers. When this does happen, however, the worker's career may be prematurely ended. Virtually all cases of transmission to patients occur when HBeAg-positive healthcare workers perform invasive procedures and cuts on their hands bleed into open lesions on patients. For example, oral surgeons frequently cut their hands on sharp instruments, wire or broken teeth, and gynecological surgeons, who cannot see their hands during

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deep abdominal surgery, frequently sustain needlestick injuries which may bleed into the patient. Transmission has also occurred when HBeAg-positive healthcare workers with exudative dermatitis performed invasive procedures without gloves.

Women who are acutely infected during the third trimester of pregnancy or who are HBeAg-positive carriers transmit the infection to the infant in 70 to 90% of births [8]. This leads to the chronic carrier state in 80 to 85% of infants infected who in turn have a 25% chance of dying of cirrhosis or liver cancer during adulthood [9]. Eighteen percent of acutely infected individuals pass the infection on to their regular sexual partners [9], and the partners of chronic carriers are almost always infected. Household contacts of chronically infected individuals are also at high risk of infection through a variety of transmission modes.

At-risk workers who make a full recovery should not be restricted from working. Those who become chronic carriers may be candidates for treatment with Interferon, which may result in significant clinical improvement, and even loss of antigen, in those for whom treatment is indicated. When treatment is not successful, the worker should be provided with individual counseling from experienced occupational health professionals or infection control specialists.

Prevention of Occupationally-acquired Hepatitis B

Defining occupational risk

The occupational risk of acquiring HBV infection cannot be determined solely by job title (such as physician, surgeon, nurse, technician) or place of work (such as operating theatre, laboratory, ward). It must take into account the tasks which expose the worker to regular physical contact with blood or other infectious body fluids.

Risk assessment

Risk assessment must take into account the possible modes of transmission -stab injury with contaminated needle or sharp instrument; contamination of broken skin or mucous membrane; or human bite [10]- as well as the frequency of exposure to contaminated sources. Four groups may be identified:

1. Healthcare professionals whose work activities involve regular physical contact with patients and/or their blood or body substances for the purpose of providing clinical care and/or investigational or therapeutic intervention for example, surgeons, nurses,laboratory technicians, dentists, paramedical staff and other allied healthcare workers such as speech therapists and dental hygienists.

2. Non-professional health care workers or support staff who are employed in healthcare or at-risk environments and have regular contact with patient's blood or body substances- for example, housekeeping and laundry workers and maintenance technicians.

3. Workers outside the healthcare setting who may have contact with blood or body substances- for example, public service workers such as police or paramedics, teachers and support workers in homes for the developmentally disabled, and embalmers and morticians. Not all members of a particular occupational group will be at equal risk, however [11]. However, persons within these groups who fulfill the definition of risk (having regular physical contact with blood or body substances) will require vaccination.

4. Students in training in the healthcare environment such as medical, dental, nursing and midwifery students. The risk to students during training may be higher than in later years when their competence increases. Many training programmes are located in urban areas serving populations with high HBV prevalence.

Risk management

Risk management is based on a comprehensive system of prevention which includes:

- * the institution of safe and consistently high standards of working practice
- * adoption of universal precautions
- * the use of appropriate personal protective equipment
- * active vaccination of those at risk
- * investigation and management of exposure incidents
- * follow-up records.

Universal precautions

Most HBV carriers are unaware of their status, nor will this be found in clinical records. The concept of universal precautions assumes that all patients may be infected with blood-borne pathogens, and stresses the importance of applying appropriate precautions to all patients and their body fluids, regardless of any diagnosis. Universal precautions also apply to decontamination procedures following blood or body substance spillage.

Universal precautions include general barrier techniques and defined procedures for the disposal of sharps. Barrier techniques should be used during contact with mucous membranes, wounds and indwelling medical equipment. These techniques may be direct (using protective clothing such as gloves) or remote (using specialist equipment).

Universal precautions apply to blood and blood products,semen, vaginal secretions, cerebrospinal fluid, pleural, pericardial or peritoneal fluid, synovial fluid, amniotic fluid, saliva in association with dentistry or oral procedures and unfixed pathological specimens.[12]

Universal precautions apply to urine, feces, vomitus, nasal secretions, sputum, sweat, and tears if they are contaminated with visible blood.

Safe working practice

The first duty of at-risk workers is to ensure their own safety by using waterproof dressings to cover any skin lesions from cuts or abrasions or conditions such as eczema on exposed parts of their body. Healthcare workers who have extensive lesions on their hands or forearms should be assessed carefully on an individual basis before participating in invasive procedures. All at-risk workers must comply with hospital and/or departmental policies on the use of personal protective clothing.

One underlying principle of safe working practice is the avoidance of 'sharps' whenever possible (13). Examples are:

- using adhesive tape, glue or staples for wound closure
- introducing minimally invasive endoscopic surgical techniques
- using lasers.
- replacing glass with plastic pipettes
- using automatic pipettes and specimen analyzers.

Sharps should never be passed hand-to-hand in operating theatres. Good surgical technique requires that needles should always be used in a needle holder, never hand-held, and the position of the point should never be checked by touch. 'No touch' techniques should be used whenever possible, appropriate protective clothing should be worn, and waste equipment and materials should be disposed of safely.

Mortuary staff and funeral personnel who may be at risk of contact with blood and body fluids must use appropriate personal protective clothing and maintain high standards of work practice. Drainage tube sites and open wounds must be covered by waterproof dressings.

Disposal of clinical waste

Sharps must be disposed of safely into approved containers, and disposable needles should not be resheathed. A risk appraisal should be undertaken to determine a method of safe disposal for larger items of 'sharp' medical equipment where the 'sharp' end cannot easily be separated or severed into a sharps container. All items contaminated with blood or body substances including soiled dressings, containers and gloves must be disposed of into appropriate receptacles (such as heavy-duty plastic bags). These can subsequently be incinerated. Workers should adhere to these guidelines both within the hospital and in the home.

Reusable medical and dental equipment that has been contaminated by blood or body substances must be disinfected and sterilized in accordance with national guidelines .

In the hospital, contaminated linen should be placed in an appropriate bag and labeled according to local procedures. In the community, blood stained linen and bedding may be washed in a domestic washing machine in the hottest cycle (approximately 80°C). Where handwashing is unavoidable, the operator must wear rubber gloves and use hot water and detergent.

Using hepatitis B vaccine in workers at risk

Effectiveness of vaccine

Safe and effective vaccines against hepatitis B have been available since 1982 and have been recommended for healthcare workers since that time. Active vaccination for those workers at occupational risk of HBV infection is an integral part of the risk management strategy for prevention of what is now recognized as the most important infectious occupational disease.

Both plasma-derived and genetically engineered vaccines are available worldwide, but the latter vaccines are used in Europe. They are highly effective, with seroconversion rates of about 95% in young healthy adults [14]. The peak antibody titre achieved following the primary vaccination series determines how long the antibody will persist at detectable levels. The generally accepted level for seroconversion and protection against infection is 10 mIU/ml [15]. The higher the initial antibody response, the longer it persists [14].

HBV vaccines are generally well tolerated and the majority of side-effects that do occur are minor and transient and are related to the site of injection; and consist of mild tenderness or local irritation comprising erythema, swelling and induration. General side effects like fatigue, headache, slight fever and gastrointestinal disturbance are uncommon, mild and self-limiting. Vaccination is not contraindicated in pregnant or lactating women. Female workers at particularly high risk of occupational infection should be advised to continue immunization while they are pregnant as the consequence of acquiring infection, both to themselves and their newborn, outweigh considerations of minor side-effects. Hepatitis B vaccines do not contain live virus particles.

Eligibility for vaccination

Eligibility for hepatitis B vaccination is determined by risk assessment of the worker's regular duties and not by job title or job location.

Schedule of vaccination

The schedules for vaccination approved by most regulatory authorities are:

- 0,1 and 6 months (first dose followed by a dose one month and six months after the first)

- 0,1,2 and 12 months (first dose followed by a dose one month, two months and 12 months after the first).

The "0,1,2, 12 months" schedule should be considered when rapid protection is required in high-risk situations. Protective antibody titres are achieved in almost all recipients by three months, and practical experience shows that compliance is significantly better within this short timescale. In order to ensure an enduring high antibody titre following rapid vaccination, a fourth dose must be given at 12 months. For routine immunization, the "0,1,6 months" schedule is less costly and provides excellent antibody titres and duration of protection.

The site of vaccination and the method of administration of the vaccine are critical factors in achieving maximal seroconversion rates. Persons vaccinated in the gluteal muscle have lower seroconversion rates and antibody titres than do those who receive all their injections intramuscularly in the deltoid muscle of the arm [16]. The needle must be long enough to penetrate subcutaneous tissues and enter the belly of the muscle, and it should be introduced at right angles to the skin surface. The intradermal route is not recommended by many expert groups [16].

Screening

A successful hepatitis B immunization programme may be implemented without pre- or post-immunization serological testing, and most community based hepatitis B vaccination programmes have no routine testing component. Hepatitis B vaccine is harmless to currently or previously infected (immune) individuals, but they receive no benefit from the vaccine.

Pre-vaccination screening using the HBsAg test, which will identify HBV carriers, is not necessary to identify previously infected individuals and may discourage participation in the programme among healthcare workers who fear job or personal discrimination if they are found to be carriers.

Many hospitals choose to test workers after immunization for one or more of the following reasons:

- to document successful immunization for medico-legal, programmatic and personal reasons
- to offer additional doses of vaccine to non-responders in an attempt to induce seroconversion
- in some hospitals, to measure titres of antibodies so that the time to booster doses may be calculated.

The potential benefits of post-vaccination testing must be weighed against potential problems. It should be kept in mind that it is successful immunization, not testing, that prevents disease; it is much more important to achieve a high level of immunization coverage in at-risk workers. Failure to vaccinate, not vaccine failure, is the primary cause of continued cases of HBV infection in health and public safety workers.

Nonresponders (those who fail to achieve an antibody titre greater than 10mIU/ml) [15] are not protected against the infection. They may respond following 1 to 3 additional doses of vaccine, but usually develop low levels of antibody.

Non-responders should be counseled carefully on safe work practices to avoid infection. Some physicians have proposed that work restrictions should be recommended for certain non-responders. If this is the case the same restriction should apply to those who choose not be vaccinated.

Booster doses

There is no published evidence that any healthcare worker who has been vaccinated successfully has subsequently developed clinical hepatitis B or the HBV carrier state. However, seroconversion to anti-HBc antibody has been reported in vaccinated populations, probably representing subclinical infection in some individuals. Antibody levels decline following immunization, dropping below 10 mIU/ml and even to undetectable levels in many individuals after 3 to 10 years. Some recommendations call for booster doses of vaccine either when antibody decays to below 10 mIU/ml or after a fixed number of years. Protocols have been developed to predict the decay of antibody and tailor booster dose timing to each individual, depending on the antibody titre attained following the primary series. The Viral Hepatitis Prevention Board has stated that a booster dose every five years is 'prudent' in workers at risk, where resources allow.

Prevention of hepatitis B in self-employed workers

Self-employed workers may not have access to occupational health services. Those who suspect they may be at risk from hepatitis B should ask their general practitioner or primary care physician for advice.

Post-exposure prophylaxis

Immediately after an exposure injury, the wound should be washed thoroughly under running water and squeezed (not sucked) to encourage bleeding. The site of injury should then be wiped with an alcohol-impregnated swab and covered with a waterproof dressing. The accident must be reported to a designated person and a written accident report filed in the worker's occupational health record. The report should name the source and the circumstances in which the injury occurred. Wherever possible, blood samples should be obtained from the source specimen or patient (with their consent) and the samples tested for HBsAg. Small samples are adequate from the very young and the very old. If permission is not granted, the patient should be assumed to be HBsAg positive. A sample of blood is also required from the worker to test for antibody status.

The results of the investigations should ideally be made available within 48 hours. This will allow the necessary treatment to begin as soon as possible. Specific hepatitis B immunoglobulin (HBIG) should be administered if the source of the injury is HBsAg- or HBeAg-positive and if the worker has not been vaccinated or has an antibody titre less than 10 mIU/ml. (It is safe to use HBIG in pregnant workers.) HBIG should be given within 48 hours of exposure to have the maximum effect [17]. If this is not possible, it could be administered up to 15 days after exposure but the effectiveness will be reduced. Even when given within 48 hours, however, HBIG is not 100% effective and workers should not rely on HBIG prophylaxis in place of vaccination.

	Hepatitis B Carrier Status of Source		
Status of expose worker	Not identified/Not available	Negative	HBsAg/HBeAg-Positive
Unvaccinated	Vaccinate + HBIG if available	Vaccinate	Vaccinate + HBIG
Vaccinated - antibody titre <10 mIU/ml at the time of accident	Booster Dose	Booster Dose	Booster Dose + HBIG
Vaccinated - antibody titre >10 mIU/ml at the time of accident	No action	No action	No action
No response to vaccine after three doses	HBIG if available +2 further vaccine doses	2 further vaccine doses	HBIG + 2 further vaccine doses
After 5 doses	HBIG if available	No action	HBIG

HBIG = Specific hepatitis B immunoglobulin

Table 1: Practical guidelines for post-exposure prophylaxis against hepatitis B

During the investigation of needlestick accidents, attempts should be made to vaccinate unprotected workers because they are at risk of hepatitis B. In those who have been exposed to HBsAg- or HBeAg-positive blood, active vaccination and passive immunization with HBIG can occur simultaneously. An accelerated course of vaccination for post-exposure prevention in those who have not been routinely included in a vaccination programme will produce effective antibody titres within the incubation period for hepatitis B. In accidents where the source is not identified, the routine use of HBIG is not recommended because of high cost, limited availability and limited effectiveness. However, if there is a high endemic prevalence of hepatitis B in the region, or if it is known that the equipment came from a unit which cares for a large number of chronic carriers of HBV, then HBIG may be indicated.

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A Case of Cushing's Syndrome

Syed Zaffar Mehdi, F.A. Bhat.

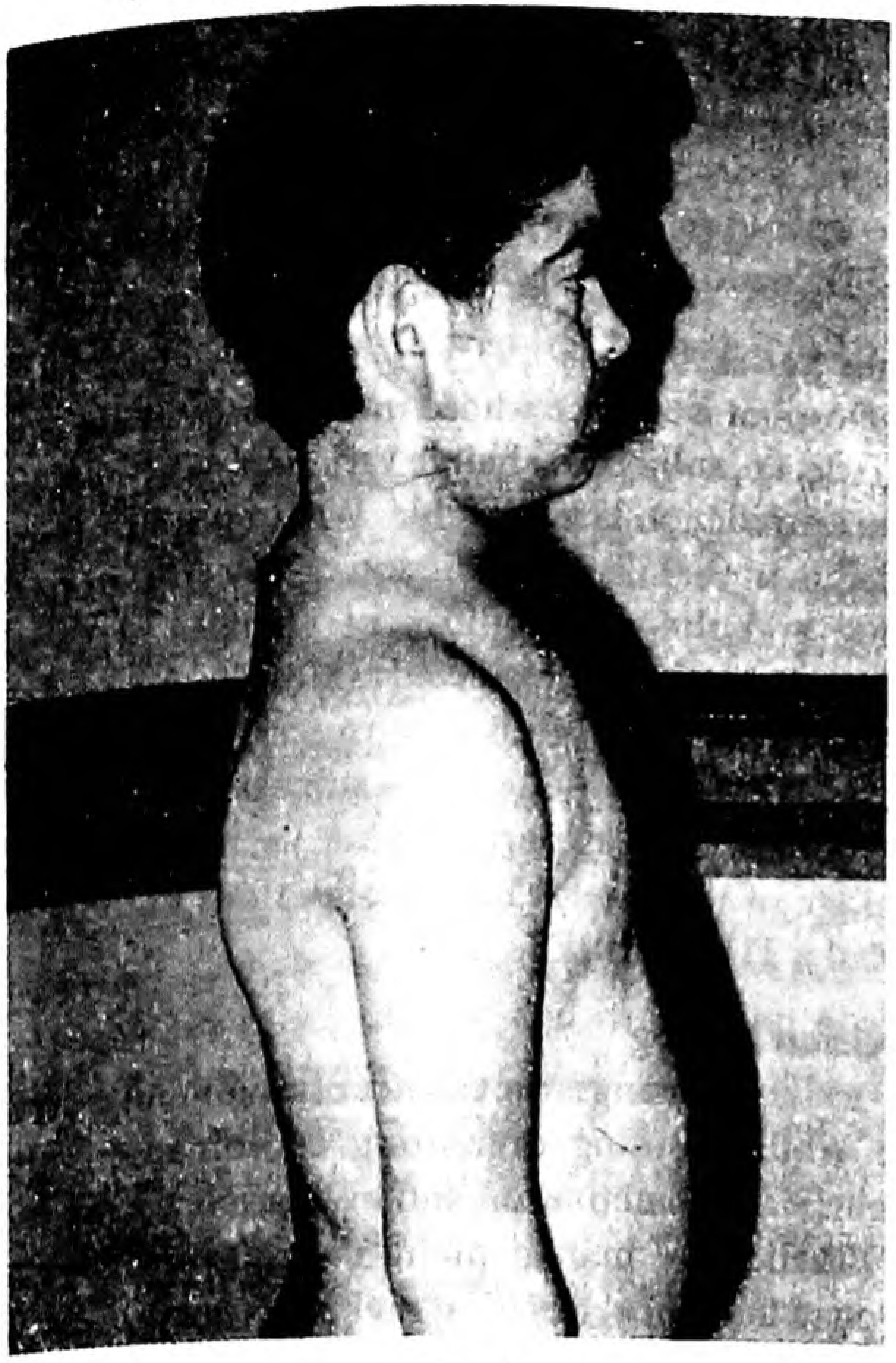

Fig. A

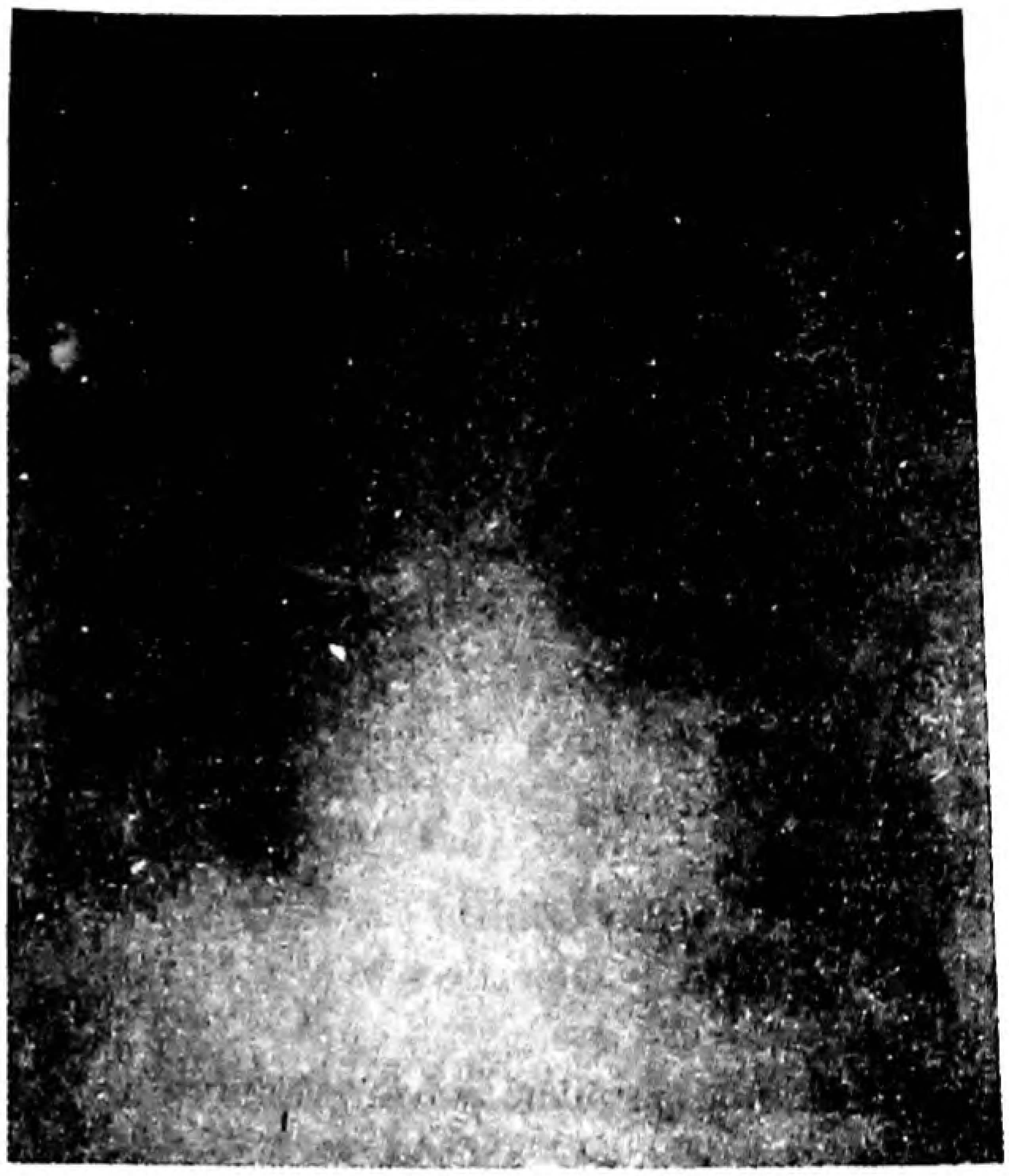

Fig. B

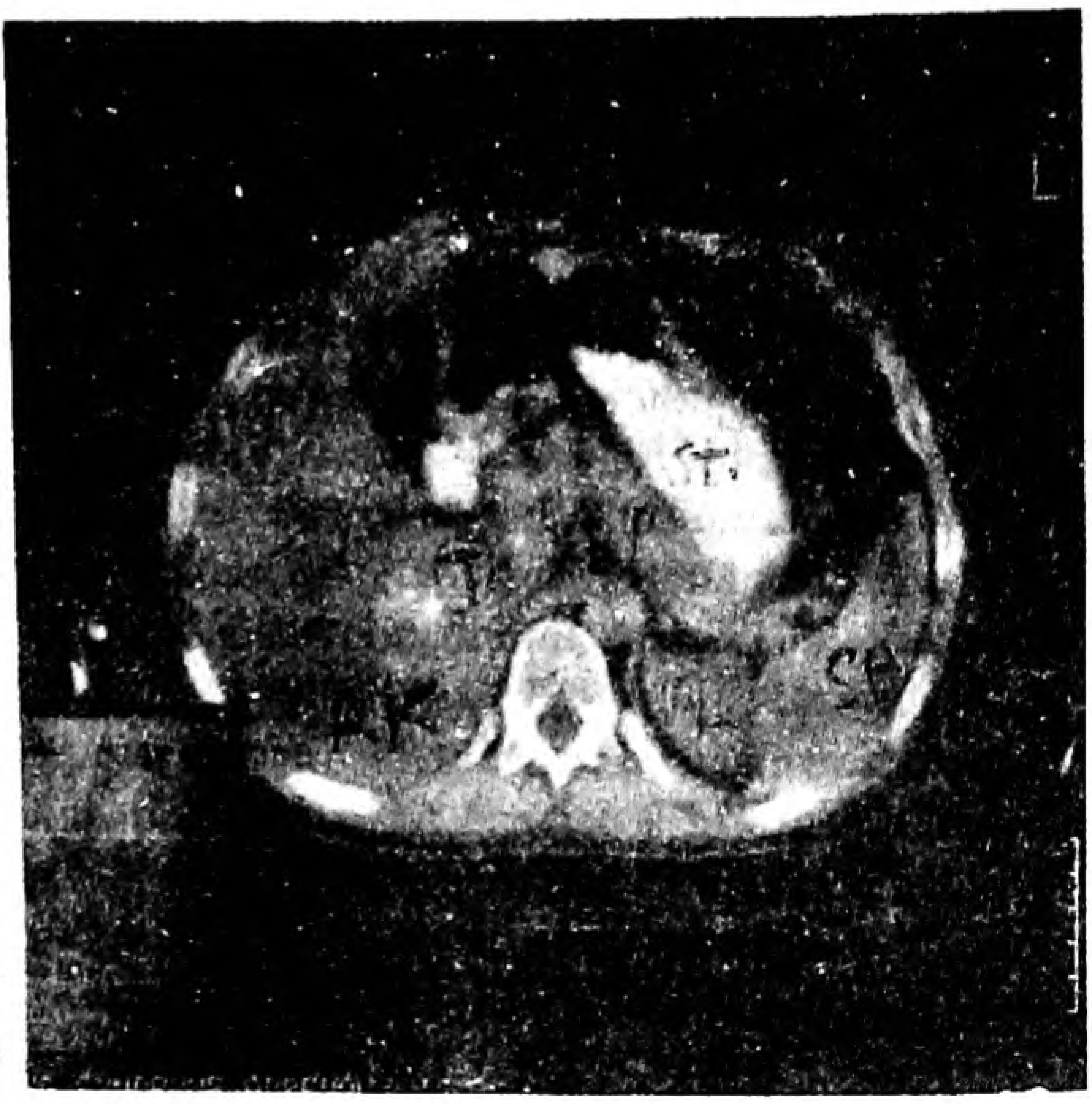

Fig. C

Fig. A: Moon facies of Cushing's Syndrome

Fig. B: X-Ray Chest showing pulmonary metastasis

Fig. C: CT Abdomen showing adrenal tumor

A 16 year old male child admitted with resistant hypertension and cushingoid features. C.T. showed an adrenal tumour. Patient died one month later due to metastasis and continued disease.

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JK Practitioner, 1995;2127-32.

Hospitals Today

Modern Techniques in Hospital Management

Syed Amin Tabish

The main function of an administrator is to take decision based on accurate data and information. There are various techniques, which are available to the manager and these can effectively be used in the field of hospital management. Hence, it is imperative that all hospital administrators have a conceptual knowledge of following managerial techniques:

1. The Feed Back Loop
2. Time Series Analysis
3. Value Analysis
4. Break-Even Analysis
5. Operations Research
6. Strategic Planning
7. Forecasting
8. Statistical Quality Control
9. Management By Objectives
10. Quality Circle
11. Management Problem Solving Methods
12. Cost Analysis
13. Utilization Management

The Feed Back Loop

The hospital, in one way, can be described as a collection of functional service units. Each of the units perform some specialized function. The departmental organizational structure of units like nursing, dietetics, radiology, laundry etc. are infact built in this manner. In a typical acute hospital there may be more than twenty such functional units. The functions of the units vary widely and these may range from removal of waste and garbage to very specialized clinical functions. Nevertheless, from the point of view of the hospital manager each of these units can represent and function a basic Feed Back loop or can provide Result Expected Information or Goal Achievement Results.

No hospital function can be effectively performed in the absence of a demand for it. Demand in a hospital is generated by process different from the functional services. The adjustment is required in various individual units, if the performance is expected at desirable level. Like a functional change of the operation Theatre would need the back up of CSSD, laundry, blood Bank etc.

Time Series Analysis

Hospital is a place where most processes are stochastic, i.e. subject to change variation. When it is so exact prediction is impossible. What must be substituted is probabilistic prediction, given an expected value and the probabilities of changes of given amounts around the expected value. The opposite of Stochastic is Deterministic, a process which always yields its expected value and is subject to no change fluctuation. The expected value i.e. the mean or average of the several historical periods in order is called a TIME SERIES.

Decision Making

Decision making is the essence of an administrator's job. While planning, organizing, coordination/controlling. implementation and evaluation are the basic functions of management, each of these clearly involves decisions. Decision making is the process of choosing between alternative courses of action. Making a decision involves four steps: (i) Identifying the central problem; (ii) Developing alternatives; (iii) Analyzing the alternatives and (iv) Making the final decision. Creativity and the effective use of groups are two important factors in developing alternatives. Most widely used alternative analysis techniques are:

a) Capital budgeting
b) Break-even analysis
c) Operations Research

Capital budgeting is used to analyze alternative capital investments.

Break Even Analysis (BEA)

Most fundamental quantitative technique available to administrators is BEA. This is a graphic representation between revenues and costs at various levels of output. The break-even point in the analysis is that point (level of output) at which total revenues will equal total costs, thus resulting in neither profit nor a loss for the organization. It indicates the quantity of output that must be achieved in order to cover total costs of operation and also the point in output at

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which profits will begin to accrue.

Break even point=Fixed costs/(Price per unit - variable per unit)

Operations Research (OR)

OR is the application of scientific methods to management problems. It refers to a set of mathematical techniques through which a variety of organizational problems can be analysed and solved. The steps in applying this scientific approach are: (i) Formulating and defining the problem; (ii) Constructing a mathematical model; (iii) Testing the model and solution derived from it and (iv) Putting the solution to work (implementation).

Specific OR Techniques include:

1. **Linear Programming:** This is a mathematical method which is used to solve resource allocation problems, i.e. optimum use of resources for achieving desired objectives.

2. **Computer Simulation:** Evaluation of alternative courses of action without direct experimentation.

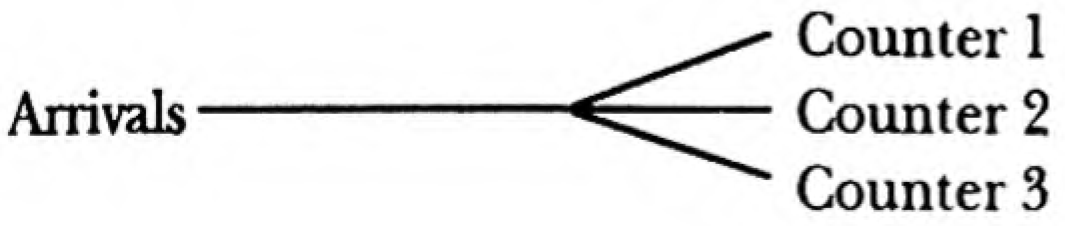
3. **PERT:** Programme Evaluation and Review Technique is a schematic model utilizing the network concept to show the sequencing of work activities, approaches to scheduling and controlling projects with reference to time and cost. In setting up a PERT network it is necessary to define the particular target. The network will then represent the series of activities and events (in proper sequence) which represent progress towards completion of the project.

4. **Inventory Model:** Problems regarding storage capacity, quantities to be ordered, and when to be ordered can be attended by using inventory models.

5. **Work Study:** Determining the standard time necessary to carry out an activity, developing of effective and easy methods of work.

6. **Queuing Techniques (Waiting Line):** Determining waiting time, cost of waiting time and its reduction, identification of bottlenecks. This technique can be used to analyze alternatives and arrive at solutions to many of these problems. It is necessary to know how often patients arrive, how long it takes to service them; and the order in which arriving patients are served with. A simple method of studying a queue line is described below:

A multiserver queue in a service area is represented as:



The following observations are made at intervals of selected unit time.

Time (hrs/min)	Unit time T	Queue length (persons waiting) Q	Number served in unit time N	Number of counters open C

Individual Service time (i.t) = TxC/N (e.t.xC)

Effective Service time (e.t) = T/N minutes

Waiting time (w.t) = Q x e.t.

Capacity (C) = 60 min/e.t.

These values depict the time spent by an individual at each counter and the capacity of the service point, which are useful in making decisions regarding allocation of time, personnel and equipment so that both idle periods and waiting time can be minimized. They also facilitate inter-service and inter-hospital comparisons.

The queuing technique can be applied in various patient care areas like Outpatient department, Radiodiagnosis and Imaging, Operations (Surgery) etc.

A) Prevalent system in OPD

Consultation chamber No.	1	2	3	4	5
Patients	1-8	9-17	18-26	27-35	36-43

B) Modified System

Consultation chamber No.	1	2	3	4	5
Patients allotted as per arrival	1	2	3	4	5
	6	7	8	9	10
	11	12	13	14	15

Strategic Planning

Planning provides direction and a sense of purpose for the organization. It also provides a unifying frame work of decision making throughout the organization. Planning helps to reveal future opportunities and threats, and also facilitates control.

The basic steps in planning include:

a) Forecast and develop planning premises
b) Define and develop planning premises
c) Define specific objectives
d) Develop and test alternatives
e) Decide on a course of action

Planning usually involves at least two phases - Strategic planning (aimed at developing the organization's basic long-term plan, or strategy), and Action Planning (short term objectives are developed for each department, short run forecasts are made, alternative action plans are developed and analysed, and specific ones are chosen).

Steps in developing a strategic plan include:

- Clarify the organization's domain, in terms of services it offers and to whom,
- Make a strategic forecast
- Determine current strengths and weaknesses
- Develop alternatives and analyze each in terms of organization's strengths, weaknesses, threats and opportunities, and
- Set specific objectives - Commence Action Planning.

The strategies of a hospital need to be well conceived, practical and commitment oriented. Strategic planning will help administrator to develop a better image for the hospital and that the hospital services will bringforth the desired results for the society.

Forecasting

It is a service whose purpose is to offer the best available basis for management and understand the implications for alternative courses of action. Forecasting provides the basic premises or assumptions on which objectives, strategies and plans are built.

Forecasting techniques include:

a) Economic forecasting
b) Time series (exponential smothing)
d) Casual
- multiple linear regression and econometrics
d) Qualitative
- judgmental
- delphi method (technological forecasting)
- marketing research.

There are three basic approaches to demand forecasting for the health care field. The first one is through historical experience. A demand which has occurred in past can be used as a basis for the future. To make the prediction more accurate the data of past several years could be reviewed after proper analysis. Time series can be used to predict based on the inference drawn from one situation or several similar situations. Like it is possible to predict the demand for a given situation in one hospital based on the demand experience of a similar hospital.

The other approach is based on Estimates of Need. Although need and demand are not synonymous, it is possible to estimate the need for hospital services by measurement of the incidence of certain diseases along with the assumption and knowledge about the type and quality of the service required to treat these diseases. If an estimate of the relationship between demand and need can be made, the expected demand can be derived from a forecast of the expected need. For example, if the expected number of births in a community is 1000 and it is known that 99% of the births occur in hospital, the expected demand for obstetrical deliveries can be forecast as 900. The third approach to demand forecasting is through direct subjective estimate of demand. At times there are situations when there is no relevant historical information on demand and no way to isolate need and its relationship to the demand. In such situations surveys of the opinions of doctors, nurses and in some cases patients provide the basis for the forecast.

Value Analysis

Value analysis is a technique to provide various types of models to management for taking decision as to which service a hospital can offer and at what cost and thus help in making decisions regarding planning the facilities and services. The hospital funds can be effectively rotated and better utilized for those services that can satisfy many patients with marginal change.

Statistical Quality Control

Hospital can generate a large number of individual statistics where each statistics is a measure of some limited aspect of quality or efficiency of the total process. An effective Information-Handling System must be able to process the incoming statistics. Almost every complex process will have some variability in the degree to which it achieves its goal. Thus, three or more observations need to be made and mean and standard deviation estimated.

The end product of the hospital is "Patient Care" and the product being intangible does not lend itself to measurement easily. Lately, four criteria have been developed which are used for measurement:-

Level of Management	ACTIVENESS OF MANAGEMENT		
	Passive : Regulatory	Service - Providing	Active : Health promoting
Lower : Functioning Short-term		NETWORK ANALYSIS	
		WORK STUDY	
		SIMULATION STUDIES	
			CAPACITY UTILIZATION STUDIES
		BEHAVIOURAL SCIENCE	
Middle : Structural Medium-term		INFORMATION SYSTEMS AND RECORD LINKAGE	
		STATISTICS AND FOQRECASTING	
		MANPOWER PLANNING	
		DEMAND MODELS	
			COST-BENEFIT ANALYYSIS
		SIMULATION STUDIES	
			ALLOCATION MODELS
			SYSTEMS ANALYSIS
Higher : Organisation Long-term		TECHNOLOGICAL FORECASTING	
		SYSTEMS ANALYSIS	
		STRATEGIC O.R.STUDIES	
		PROGRAMME BUDGETING	
			COST-BENEFIT ANALYSIS
		MANPOWER PLANNING	
	STATISTICAL TRENDS		

Table 1. : Application of Different Techniques according to their level of formality and to the level of Activeness of Management.

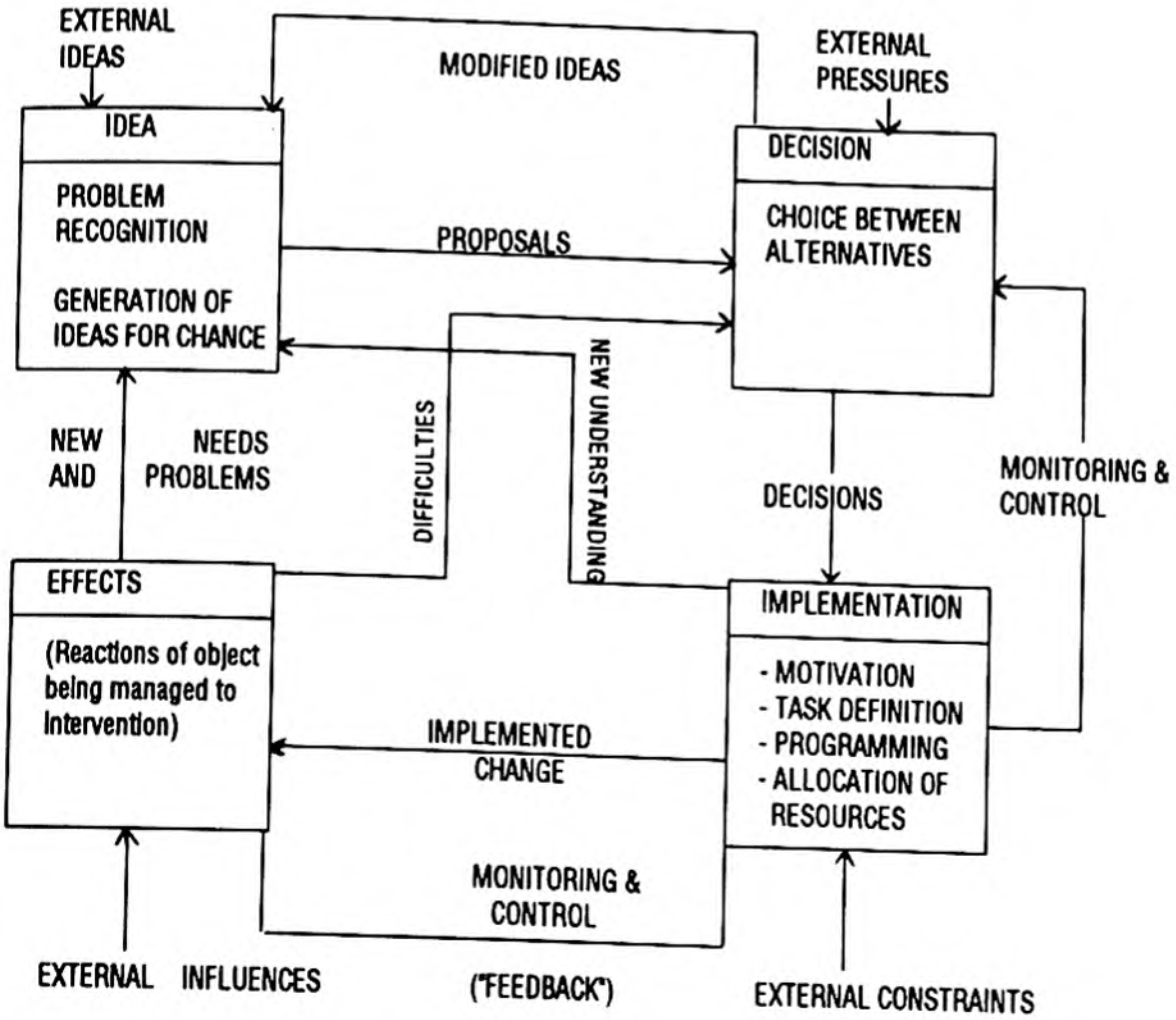

EXTERNAL IDEAS
MODIFIED IDEAS
EXTERNAL PRESSURES
IDEA
PROBLEM RECOGNITION
GENERATION OF IDEAS FOR CHANCE
PROPOSALS
DECISION
CHOICE BETWEEN ALTERNATIVES
NEW UNDERSTANDING
DIFFICULTIES
NEW AND NEEDS PROBLEMS
DECISIONS
MONITORING & CONTROL
EFFECTS
(Reactions of object being managed to Intervention)
IMPLEMENTATION
- MOTIVATION
- TASK DEFINITION
- PROGRAMMING
- ALLOCATION OF RESOURCES
IMPLEMENTED CHANGE
MONITORING & CONTROL
EXTERNAL INFLUENCES
("FEEDBACK")
EXTERNAL CONSTRAINTS

FIG. 1. The Management Process

Patient care	No. of OPD cases No. of admissions No. of operations No. of tests
Quality	Patients cured/Improved Infection/Complication
Cost	Per day per patient (in patient/out patient) Patient's satisfaction Random patient opinion/ survey staff satisfaction etc.

Management by Objectives (MBO)

MBO is a process whereby the superior and the subordinate managers of an organization jointly identify its common goals, define each individual's major areas of responsibility in terms of the results expected of him, and use these measures as guides for operating the unit and assessing the contributions of each of its members. The concept rests on a philosophy of management that emphasizes an integration between external control (by managers) and self-control (by subordinates). The smooth functioning of this system is an agreement between a manager and subordinate about that subordinates own or group performance goals during a stated time period. The goals are jointly established and agreed upon in advance. This is then followed by a MUTUAL review of subordinate's performance in relation to accepted goals at the end of the time period.

Throughout the time period, what is to be accomplished by the entire organization should be compared with what is being accomplished; necessary adjustments made and inappropriate goals discarded. At the end of the time period, a final mutual review of the objectives and performance takes place. If there is a discrepancy between the two, efforts are initiated to determine what steps can be taken to overcome the problem. This sets the stage for the determination of objectives for the next time period.

Quality Circle (QC)

QC is a people building philosophy capable of providing astonishing results. QC is a small group of people doing similar or related work who meet regularly to identify analyze and solve quality, productivity, cost reduction, safety and other problems in their work area, leading to improvement in their performance and enrichment of their work life. Membership is voluntary (6-12 members) meet on an average one hour one a week. The objectives of QC include: (i) cost reduction; (ii) reducing employee turnover; (iii) improving employee morale; (iv) greater efficiency of operations; (v) more effective scheduling; (vi) reduce absenteeism; (vii) improve nurse-clinician relations; and (viii) more accurate cost-allocations;

Significance of QC lies in utilizing the education, experience and creativity of the staff in hospital problem solving and decision making. QC is an integrated programme made up of: (i) the members themselves; (ii) circle leaders; (iii) the facilitator; (iv) the co-ordinator; and (v) steering committee (group of key individuals selected from management, circle leaders and members of all major functional areas).

The process include identification, selection and analysis of the problem followed by implementation and recommendation to the management.

Implementation involves

- * Establish atmosphere
- * Obtain commitment
- * Select objective
- * Inform
- * Voluntary
- * Training
- * Be open and positive.

The key success is a good implementation plan.

Basic techniques of problem solving

(i) Brain storming; (ii) Data collection; (iii) Fishbone (cause and effect) diagram; (iv) Linear graphs; (v) Frequency distribution; (vi) Scatter diagram; (vii) Histogram; and (viii) Prrete analysis (drawn as a column graph)- identification of the few from trivial many.

The concept of QC envisages participatory management style with a "Bottom-Up" approach.

Management Problem Solving Methods (MAPS)

The organizational ability can be improved by the use of MAPS. The problem solving, the case study approach, involving actual or contingency situation is suitable for all management levels.

Units of Analysis

1. Overall organization
2. Resources flow
   - Personnel
   - Funds
   - Equipment information flow

Types of patients (infection rates, medication errors, patient waiting times)

3. Work group
4. Organizational environment. (including social, economic and technological factors)

With the analysis of data provided in these categories the organization unit has available performance criteria which can be used to illustrate the percentage success in providing a service to the patient. This will show how efficiently organized the hospital is in providing a service to the patient. MAPS can be applied to organize hospital equipment maintenance.

Cost Analysis

The growing complexity of hospital operation in size and volume, increase in demand of services and high standard of care requires systematic use of hospital accounting system in general and cost accounting in particular.

The process of cost analysis has become a resource tool for financial management in hospitals. The cost of operating different services are located to different cost centers. Costing also helps to assess the efficiency and effectiveness of functions and their cost implications with a view to contain cost. The exercise of unit costing involves the determination of the cost of single unit of any product or service. The approaches to unit costing are:

1. Standard costs (gives an estimate of what the costs should be i.e. it accumulates the costs incurred for all material and labour component of a product/service and provides allowance for any normal wastage. Scientific methods like time and motion studies can be used to compute the cost).
2. Average costs (indicates the actual average cost of providing one unit of service. This is a top down approach, where the total costs of the service center are determined and divided in a rational proportion among the different services offered- thereby deducing the cost of providing a unit of service. The wastage component cannot be determined by this method and hence there is no comparison standard).

Utilization Managment

It is a hospitalwide multidisciplinary programme which helps a hospital to achieve its objectives for cost control and overall institutional management. It is a catalyst for coordinating quality assurance, utilization review and risk management activities. Costeffectiveness is accomplished through the judicious use of resources to control inappropriate in patient admissions, length of stay and use of ancillary services. Utilization review is the process of assessing medical care to ensure its quality, medical necessity, and appropriateness in terms of level of care and treatment setting. It has emerged as a key feature of cost management.

Value improvement process is a proprietary management consulting technology designed to help hospital's budget. It locates opportunities for cost saving and reengineering health care delivery systems. it produces valuable cost reduction and quality improvements.

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Case Report

Blindness following Snake bite: A case report and review of literature

*S.P. Gupta, Anil Arora, Nikhil Tandon, Radhika Kunte**

Abstract

A rare and an interesting complication of blindness due to optic neuritis and vitreous hemorrhage following snake bite poisoning was observed and successfully treated. A survey of the literature revealed only a few such cases. Possible mechanism of blindness is discussed. JK Practitioner 1995; 2:133-4.

Intorduction

Snake bite is a common problem in the tropics especially in rural areas. Though most of these are non poisonous, a number of systemic complications, like neurological, hematological and renal have been reported following bites by poisonous snakes. Ophthalmological complications are usually secondary to hemorrhagic complications in the brain but primary eye involvement, though rare has also been reported [1-7]. We describe herewith a case of snake bite poisoning who developed optic neuritis and vitreous hemorrhage, and was treated successfully.

Case Report

A 20 years old male was hospitalized in a comatosed state three days after a snake bite on his left foot. He was asymptomatic till one day prior to his admission when he complained of severe headache and vomiting following which he lost consciousness.

On examination he was in grade III coma. Pulse rate was 52 per minute regular, blood pressure 130/80 mmHg and temperature 38°C. Neck stiffness was present. There were two fang puncture marks on the dorsum of his left third toe without any appreciable local reaction. Two large ecchymotic patches were found on his right arm. Both pupils were widely dilated and reacted sluggishly to light. Direct ophthalmoscopy revealed disc hyperemia with blurred margins with two peripapillary hemorrhages in right eye. There was mild venous engorgement and spontaneous venous pulsations were present. Examination of the left eye showed evidence of vitreous hemorrhage. Fundus details were not visible.

There was no focal neurological deficit and plantar reflexes were flexor. Examination of the abdomen, chest and cardiovascular systems revealed no abnormality. Investigations at the time of admission revealed Hb 12.0 gms%, TLC 8200/cmm with normal differential counts and a platelet count of 1,80,000/cmm, Bleeding time 5 mts; clotting time showed blood to be incoagulable. Urine examination did not reveal any proteinuria or microscopic hematuria. Blood urea 10.34 mmol/l (Normal=3.0-6.5 mmol/l), serum creatinine 70.64 umol/l (Normal=50-100 umol/l), SGOT/SGPT 0.44 kat/L (Normal = 0-0.58)/0.64 kat/L (Normal = 0 - 0.58), and a normal skiagram of chest. CT scan of brain revealed hemorrhage in left parietal region and in lateral ventricles. A diagnosis of snake bite envenomation with intracerebral, subarachnoid and left vitreous hemorrhage with a possible bilateral optic neuritis was made.

The patient was treated with 100 ml of polyvalent antivenom serum (Haffkin Institute, India), 0.5 ml of tetanus toxoid and corticosteroids to reduce the cerebral edema. Within 36 hours of giving antivenom serum his coagulation profile returned to normal. Clotting time was 8 minutes and tests for disseminated intravascular coagulation were negative. He became fully conscious within four days of instituting corticosteroid therapy.

On regaining consciousness he complained of blindness in both eyes. In right eye vision was limited to counting fingers at a distance of one foot. In the left eye he had only light perception and projection of rays was inaccurate in all quadrants. The pupils remained dilated with sluggish reaction to light. Fundoscopy showed no change from the earlier examination.

In one month's time visual acuity in right eye had improved to 6/12 and in left eye projection of rays had become accurate. The vitreous hemorrhage in left eye had not cleared up. Visual field examination of right eye revealed a concentric contraction in both central and peripheral fields. Pattern reversal visual evoked response in right eye showed a marked increase in latency and diminished amplitude and no

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potential generation in the left eye thus confirming a diagnosis of optic neuritis.

Discussion

Systemic envenomation following snake bite may cause cardiotoxicity, hematotoxicity or neurotoxicity. Our patient had hemorrhagic complications. In addition he had two other, relatively rare,ocular complications in the form of bilateral optic neuritis and vitreous hemorrhage in left eye.

Optic neuritis following snake bite is rare and there are only a few reported cases in world literature [1-7]. The cause of this complication remains obscure. It has been postulated that exsanguinating hemorrhage leading to acute hypotension and decreased optic disc perfusion is responsible [3]. Another theory ascribes it to demyelination caused by the antivenom serum [2]. A third hypothesis postulated is that it may be due to a direct toxic effect of some component of the snake venom. Vengal reported two patients who developed optic neuritis after being bitten by the same snake in succession [5].

In our patient, the diagnosis of optic neuritis was confirmed (by visual evoked responses) only on his regaining consciousness but he seemed to have optic neuritis and vitreous hemorrhage at the time of admission prior to the administration of antivenom serum as evidenced by presence of widely dilated sluggishly reacting pupils, hyperemic disc with blurred margins and preserved venous pulsations. Also as there was no major overt hemorrhage or acute hypotension, we presume that optic neuritis in our case was probably due to direct action of snake venom. His visual recovery may have been assisted by the administration of systemic steroids.

The other interesting feature in our patient was the vitreous hemorrhage in left eye. We could come across only one previous report of vitreous hemorrhage following snake bite. This could be a part of the hemorrhagic complications leading to retinal hemorrhage percolating into the vitreous or due to subarachnoid hemorrhage extending into the eye which is well known as Terson's syndrome. Since this patient had unilateral hemorrhage the former cause is more likely.

We conclude that optic neuritis and vitreous hemorrhage are rare complications of snake envenomation. Early diagnosis and prompt therapy may result in marked improvement.

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Young Scientist Award

Be it known that **Dr. G. N. Yattoo**, Consultant, Department of Gastroenterology, S. K. Institute of Medical Sciences, Srinagar (Kashmir) has been awarded the "**Young Scientist Award**" of the Jammu and Kashmir State Council for Science and Technology for his contribution in the field of Medical Sciences for the year 1994. His work in the field of gastroenterology and hepatology has been adjudged the best during the year.

Dr. Yattoo graduated from Medical College, Srinagar and did his post-graduation (MD, General Medicine) from S. K. Institute of Medical Sciences, Soura, Srinagar.

Case Report

Lupus Erythematosus Profundus (Panniculitis)

Qazi Masood, Sheikh Manzoor

Abstract

Two cases of lupus erythematosus profundus (panniculitis) of two females having lesions limited to the face are presented. This is an unusual clinical variety of lupus erythematosus (L.E), in which the cutaneous infiltrate occurs primarily (though not exclusively) in deeper portions of the corium with only microscopic epidermal changes giving rise to firm sharply defined nodules from 1 to several centimeters in diameter [1]. The overlying skin may appear normal or it may be red, atrophic, poikilodermatotic, hyperkeratotic or ulcerated. They can develop during the course of SLE or can be the presenting feature.JK Practitioner 1995; 2:135-6.

Case Report

Case 1 (Fig 1).

A 32 year old married, child bearing female presented with disfiguring lesions over both sides of the face and forehead of 6 years duration. Initially lesions started with small nodulelike structures which progressively increased in size. The physical examination revealed well demarcated infiltrative lesions which were firm in consistency on both sides of the cheek and on the forehead. The overlying skin of forehead lesion was normal, but that over cheek lesions showed mild central scaling with brownish discoloration and slight atrophy in the center. There was an erythematous hue at places. There was no otherclinical sign present related to any other system. Investigations revealed a normal hemogram with a normal erythocytic sedimentation rate, normal renal parameters, X-ray chest and ECG. LE cells and latex agglutination tests were negative. ANA was positive. Aspiration biopsy showed only lymphocytic infiltration. Biopsy of the lesion revealed epidermal atrophy, hydrophic degeneration of the basal layer, and infiltration of lymphocytes around the appendages. The lower epidermis & subcutis showed necrobiotic changes,,vasculitic features and cellular infiltration.

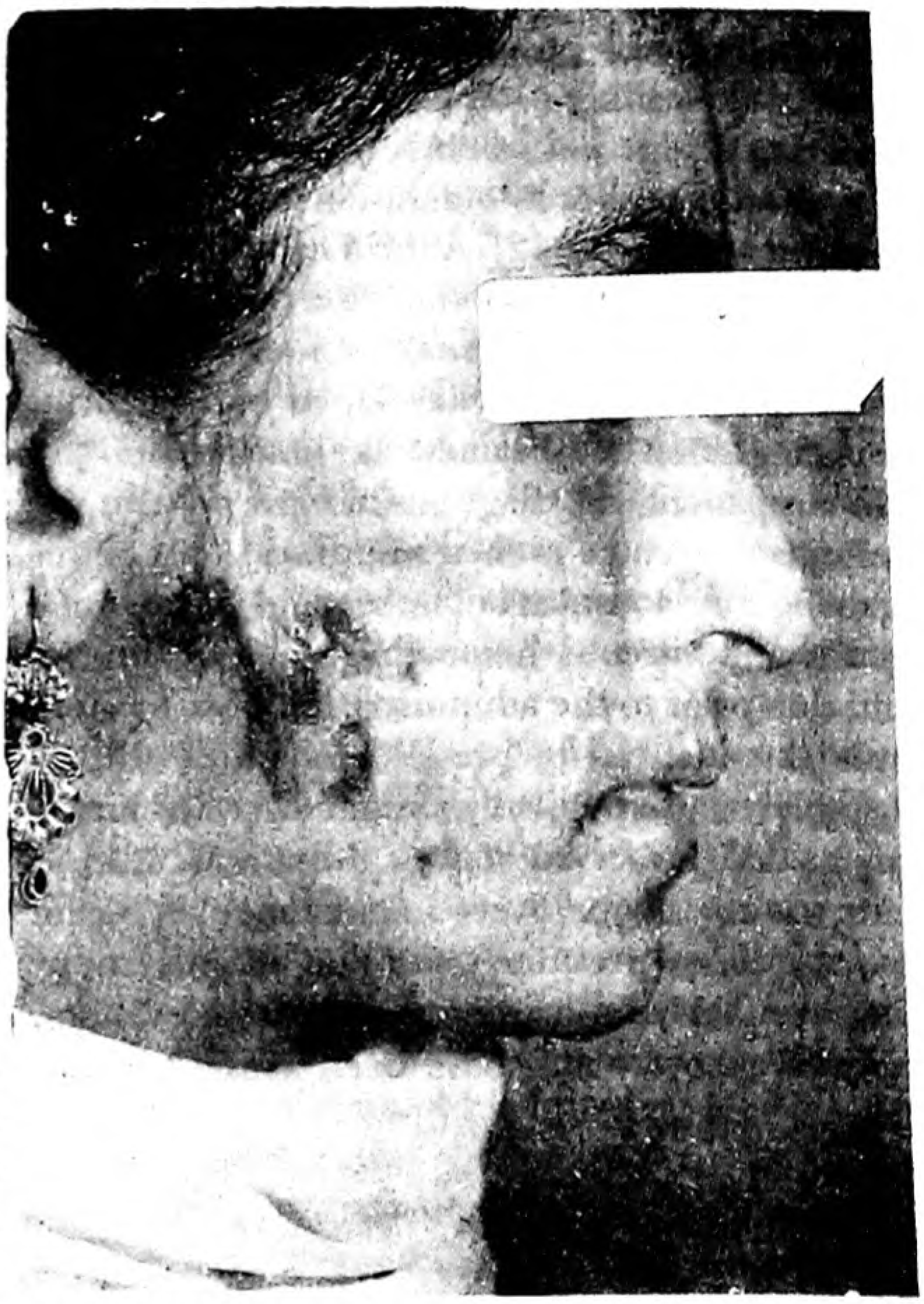

Fig. 1: Facial Lesion

Case 2

A 25 year old unmarried girl came with the complaints of firm nodular lesions over face of 3 years duration, for which she had received various treatments without any response. The lesions were progressing in size without any accompanying involvement of other organs. Her examination revealed a healthy looking girl with well demarcated firm nodular lesion over both sides of the face. Systemic examination did not reveal any other abnormality. Her investigation revealed a normal hemogram, ECG, X-ray chest and renal parameters. ANA was positive, but latex agglutination and LE cell phenomenon were negative. Biopsy was consistent with lupus erythematosus profundus.

Discussion

Lupus erythematosus profundus (Panniculitis) is an unusual clinical variety of LE in which the cutaneous infiltrate occurs primarily (though not exclusively) in deeper portion of the corium, with only

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microscopic epidermal changes, giving rise to firm sharply defined nodules. The subcutaneous nodules in LE were first described by Kaposi in 1883 (2), but the term "lupus erythematous profundus" was first used by Irgang in 1940 (3). Some others have considered the lesions to be sarcoid, but it is now usually accepted as a variant of L.E. (4). Clinically the nodular lesions are of varying size, but are usually from one to several centimeters in diameter. They are usually firm, rubbery sharply defined and persistent, seen most frequently on the cheeks but can occur on the face, arms, hands, breasts, buttocks, trunk or legs. Healing usually leads to the development of depressed scars and rarely to soft slightly pink areas of autoderma, upto 4 cms in diameter. Calcification can occur and be extensive in the extrusion of thick yellowish material, through the ulcerated areas. The nodules in the cheek may lead to marked disfigurement.

Microscopically, there may be epidermal atrophy, hydrophic degeneration of the basal layer, follicular plugging and necrosis of the dermal collagen suggesting LE,and collection of lymphocytes occurs.

In our two cases the lesions were clinically and histologically lupus erythematosus profundus, and were limited to face without any systemic features of lupus erythematosus. Both the patients were given local infiltration with steroids to which they showed good response. Both are under our follow up to see for development of any systemic manifestation of collagen vascular disease.

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Case Report

Myocardial Infarction Precipitated by Treadmill Exercise Test

Khurshid Iqbal, Nisar Ahmad, S. Jalal, B.A. Wani

Abstract

The occurrence of acute myocardial infarction, a rare complication of exercise stress testing is described in a male of 50 years with a brief review of literature. This patient developed an acute inferior wall myocardial infarction shortly after performance of a multistage exercise test. JK Practitioner 1995; 2(2):

Introduction

Exercise stress tests are widely used in diagnostic and prognostic evaluation of patients with known or suspected cardiac disease especially in coronary artery disease (CAD). Doan et al [1] emphasized the value of exercise testing in detection of ischemia in asymptomatic men with a sensitivity of maximal exercise test nine times greater than that of the double Master's two step test [2]. Serious complications attributable to stress testing as reported in the literature are rare. Rochmis and Blackburn [3], reported 16 deaths out of a total of 170,000 tests, 3 out of 10,000 required hospitalization within a week of the performance of exercise test. Myocardial infarction was considered a rare complication.

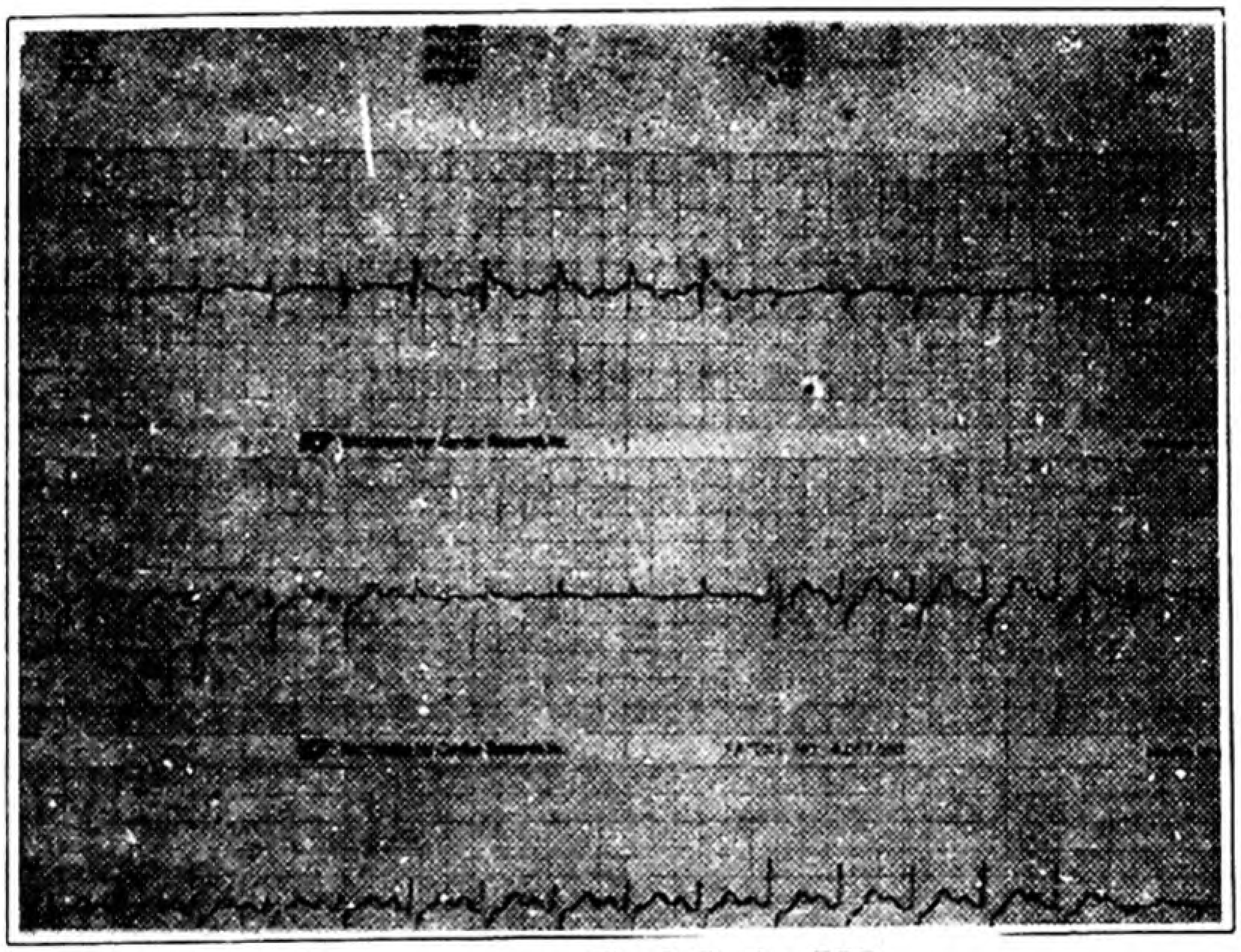

Fig. 1: Resting ECG

Presentation of the case

BMM, a 50 years male was admitted to cardiology ward to exclude ischaemic heart disease. His main complaint was difficulty in walking without support for the past six months. There was no history of chest pain, syncope or exertional dyspnea. He was not a known hypertensive or diabetic. For the present complaints he was receiving tranquilizers. One year previously patient was admitted in another hospital as a case of suspected myocardial infarction. The electrocardiogram was reported inconclusive and the patient was discharged on calcium channel blockers and nitrates without any benefit.

On examination, he was of average build, looking anxious. Pulse was 80 per minute, regular and blood pressure 120/80mmHg. Examination of cardiovascular system was unremarkable. Chest was clear and neurologic evaluation did not reveal any deficit.

Investigations including complete serum chemistry, hemogram and urinalysis were normal. Chest X-ray showed normal cardiac size and lung parenchyma. Basal electrocardiogram (Fig. 1) revealed sinus rhythm. left anterior fascicular block, non-progression of R wave in precordial leads with a QS pattern in V_5 & V_6 and T wave flattening in AVL suggestive of remote anterolateral infarct.

An exercise treadmill test was carried out using ICR documentor. The patient was able to complete three stages of modified Bruce protocol achieving a maximum heart rate of 172 per minute with no ST-T changes. The test was terminated after 9 minutes of exercise as patient complained of persistent chest pain, shortness of breath and fatigue. Fifteen minutes and 26 seconds post-exercise, uplooping ST depression became discernible in anterior chest leads with increase in the R wave amplitude in the lead V_5. This persisted till 20 minutes and the patient was shifted to CCU for further management. The ECG taken in the CCU (Fig. 2) revealed "hyperacute"

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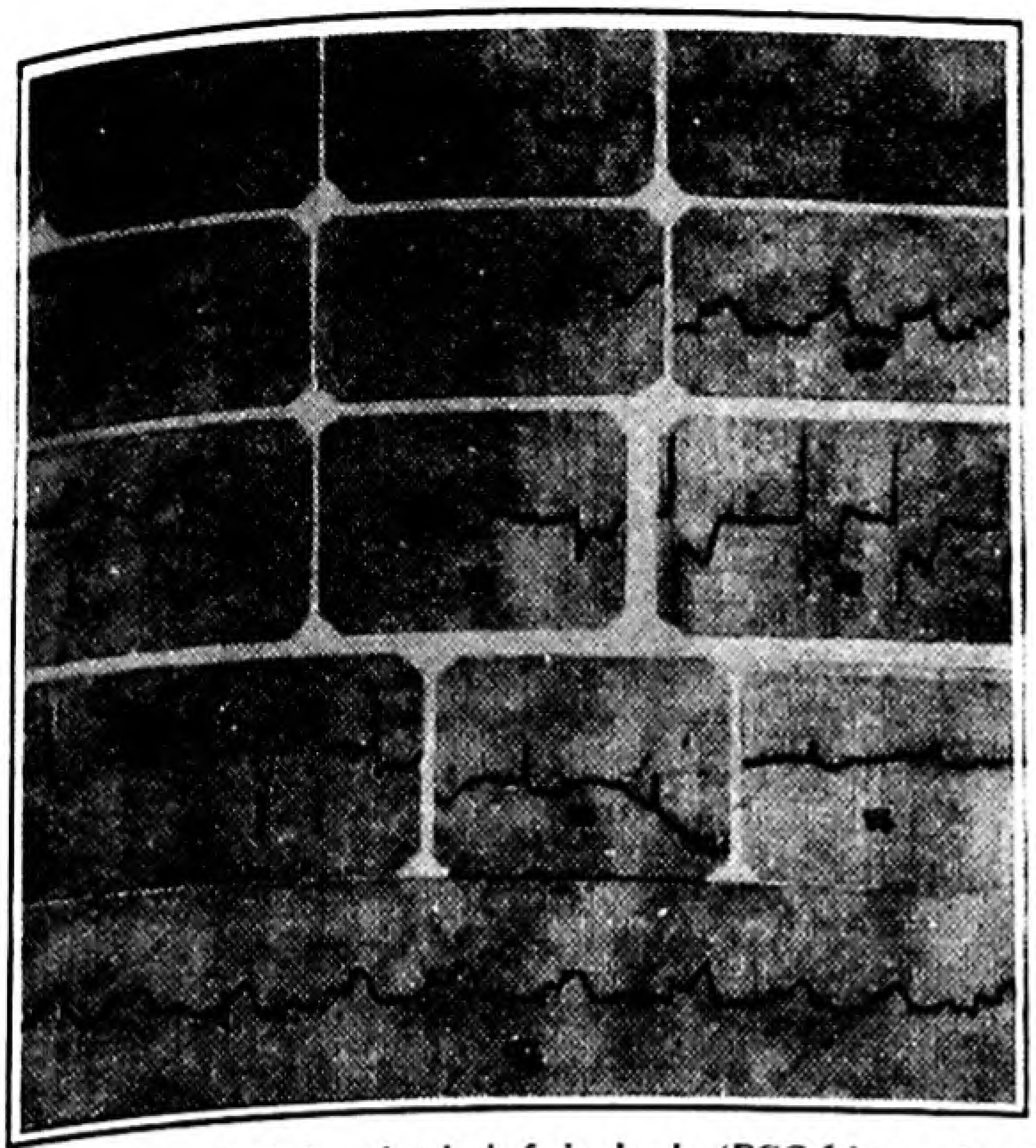

Fig. 2: Marked ST elevation in inferior leads. (ECG 1 hour after the test)

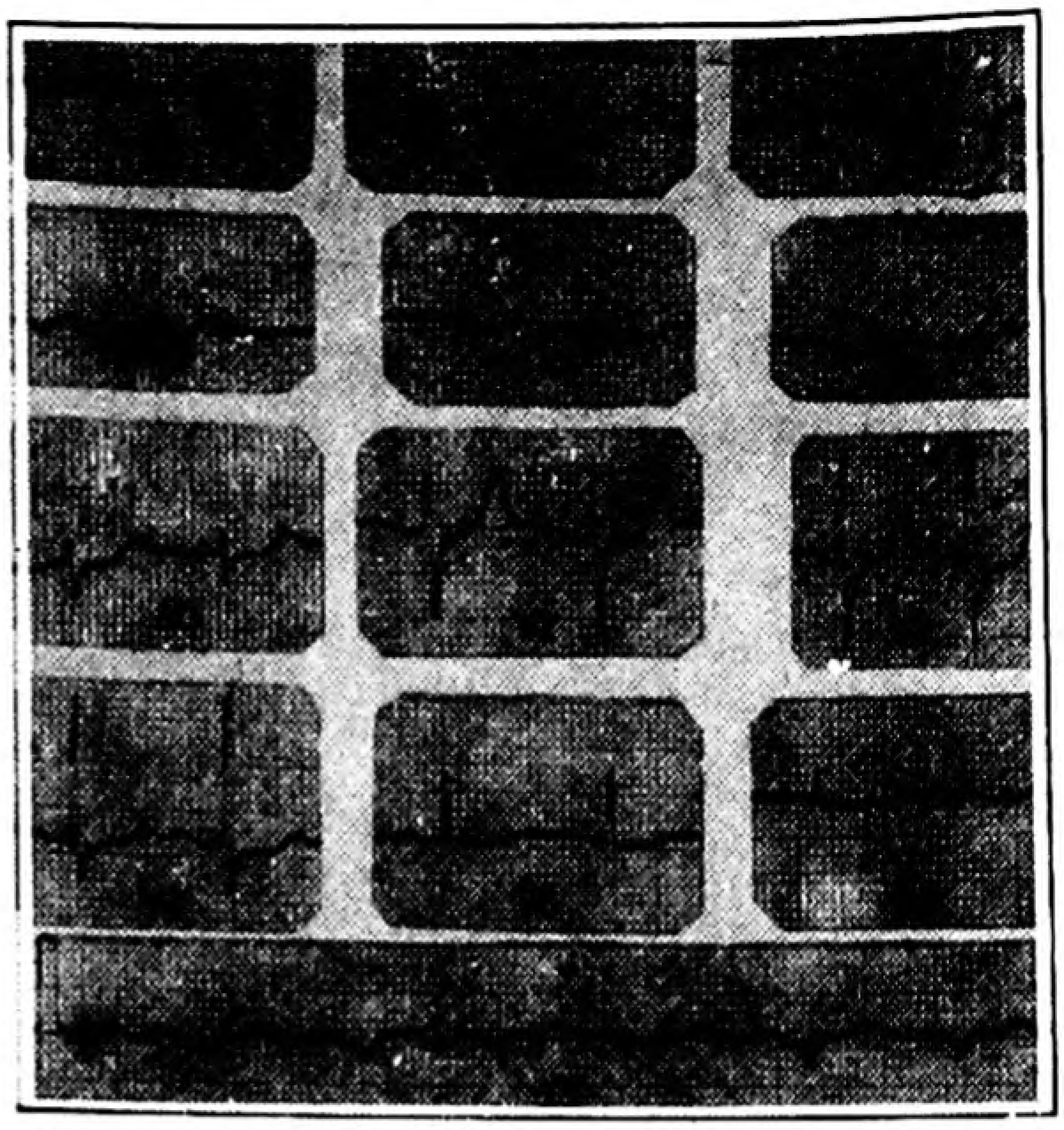

Fig. 3: Fully evolved inferior wall MI. (ECG 5 hours after the test)

inferior myocardial infarction with marked ST depression in the chest leads and Mobitz type-I second degree AV block. Shortly thereafter patient's BP fell to 90mmHg systolic accompanied by severe chest pain which was controlled by injectable meperidine. Five hours later the ECG (Fig. 3) showed Q waves in leads II, III & AVF and lesser ST depression in chest leads. He had marked elevation of CPK. Subsequent hospital course was uneventful.

Discussion

Our patient was admitted with no cardiac complaints but had a past history of a possible myocardial infarction. There was no contraindication to exercise testing. The patient suffered acute inferior MI soon after the performance of a multistage exercise test. Acute MI precipitated by exercise stress test was possibly first reported by Grossman et al [4], way back in 1955, in a patient with angina pectoris. The patient could make 8 trips over the stairs after which the test was stopped because of unusual dyspnea. The ECG revealed acute anterior MI. Bruce et al [5], reported a case of anterior MI soon after taking a shower after completion of the test with normal response to maximal exercise.

In a prospective study of complication rates in 50,000 tests, Atterhog et al [6] reported an incidence of 1.4 infarcts, overall mortality of 0.4 and morbidity of 5.2 per 10,000 tests with greatest risk in patients with aortic stenosis. Thus exercise tests may be entirely safe if patient selection and monitoring during testing is adequate.

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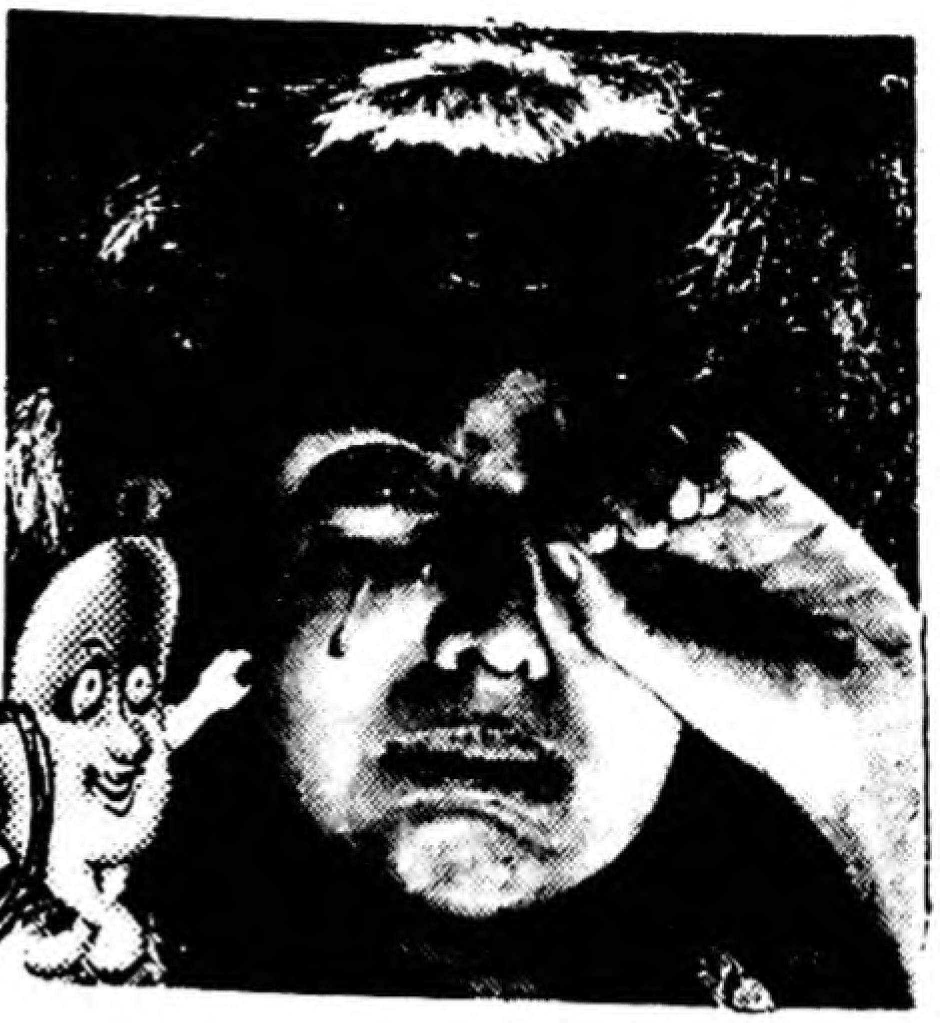

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One year of JK Practitioner

It is a great pleasure for us to complete one year of *JK-Practitioner* in October, 1995. This journal was released with the aim to help continuous medical education in the state especially in the remote areas, and to provide an impetus to quality medical research. With the help and advice of senior colleagues here and abroad (especially Kashmiri born scientists) our editorial board has succeeded to publish 5 issues covering wide ranging topics in its editorials, recent advances (updates), original articles, case reports, clinical images etc., in all specialities of medicine. We are happy that it has helped and guided our young doctors to publish their material, based on local experience and we have been encouraged by the overwhelming response the journal has had from rest of India and abroad, in the form of quality contributions. Each article is subjected to a single or double peer review before editorial revision, in keeping with the international standards. The process has resulted in publication of articles for which we have received acclaim from all over the world. We take this opportunity to thank our contributors and reviewers. As a mark of respect to our brilliant teachers who have served here or were trained here but serve abroad, *"Meet the Professor"* column was introduced, and has generated a lot of excitement amongst the youngsters when they were introduced to the sons of the soil who have made their mark at the international level. Notable among these is Prof. Faroque Khan who is heading Medicine department in New York. More such brilliant scientists of this soil will be introduced in future. A special page as obituary/memoriam has been added in memory of our beloved teachers and colleagues. The deficiencies of the previous issues have largely been taken care of in the last published issue (July-Sep,1995), wherein the editorial board went ahead with a total overhaul of the format. We are sure that the pending shortcomings shall be addressed in the future issues, and under continuous patronage of our colleagues from here and abroad the journal shall soon be able to stand with various international journals of repute. Efforts are on to get the journal cited in various *On-line* services and the *Index Medicus*. . 300 copies of the journal are sent free of cost to distant hospitals of the state. We realize that the practice of medicine is under constant evolution and that some of the opinions expressed may be subject to discussion. We would welcome our readers' thoughts on such matters. We are sure that our readers shall continue patronizing us through their contributions and valuable suggestions.

G.M.Malik
Editor in Chief

Editorials

Polyangiitis overlap Syndrome

(JK Practitioner 1995;2:142-4)

Vasculitis is an inflammation and necrosis of blood vessels leading to a compromise of the vessel lumen and, ultimately, ischemia of the organs supplied by these vessels. On the basis of their characteristic clinical and pathologic features, distinct categories of vasculitic disorders have been defined [1]. However many patients with systemic vasculitis manifest clinicopathologic characteristics which do not fit into any classification, but have overlapping features of classic polyarteritis nodosa, allergic angiitis and granulomatosis, Wegener's granulomatosis (WG), Takayashus' arteritis or the hypersensitivity group of vasculitis. This subgroup has been termed as the "polyangiitis overlap syndrome (POS)" [2]. Bradley et al [3] described a patient with temporal arteritis and pulmonary granulomatous vasculitis which possibly represented an overlap between WG and temporal arteritis, and a group of 10 cases of POS were subsequently reported by Leavit & Fauci [4]. A reappraisal of previously described cases identified many cases, that could be classified as POS. These included those reported as visceral giant cell arteritis [5,6] polyarteritis nodosa associated with pulmonary hemorrhage [7] leukocytoclastic vasculitis involving the skin plus polyarteritis nodosa [8], isolated granulomatous vasculitis involving the colon [9] and temporal arteritis associated with pulmonary granulomatous vasculitis [3]. In this issue of *JK practitioner*(see page 184), a case of POS has been reported by Haji et al which had overlapping features of Churg Strauss Syndrome (CSS) and WG.

POS is characterized by a necrotizing vasculitis of medium and small sized leukocytoclastic vasculitis, granulomatous inflammation or lung involvement; and not fulfilling the criteria for one of the other recognized vasculitic syndromes [4,10]. By using combined clinical, serologic and histopathologic criteria for classification, most patients with POS can be classified as having (incomplete form of) WG or CSS [10]. The syndrome of POS is not uncommon and a good number are initially diagnosed as a one of the classic vasculitides; POS is seen in a younger group of patients than the group with systemic necrotic vasculitis (mean age of onset being around 25 years as against a mean age of 41 years in classic necrotizing systemic vasculitis) [4]. POS can involve multiple organ systems and is indeed a systemic vasculitis. The organ involvement includes that of the skin, kidney, lung, muscle, gastrointestinal tract, joints, heart, peripheral nervous system, aortic arch and the liver with overlapping clinicopathologic features [4,11]. Cutaneous involvement, observed to be the most common organ involvement, may be in the form of either a leukocytoclastic venulitis or an arteritis [4], and is responsive to treatment as compared to a frequently unresponsive isolated cutaneous vasculitis [12]. Nearly all patients present with constitutional symptoms and rapidly progressive systemic disease. Neurologic manifestations are frequently found and involvement of the peripheral nervous system is often a dominating feature at the beginning of the disease [11]. Angiographically documented central nervous system involvement as part of POS has not been observed, which might be related to the rare occurrence of either biopsy proved angiographically documented central nervous system disease in systemic vasculitic syndromes in general [1,13].

Respiratory tract involvement is frequently observed in the granulomatous pulmonary vasculitides of WG and CSS. While asthma with diffuse patchy infiltrates dominate the clinical picture in CSS [14], upper respiratory tract involvement in the form of nasal sinus mucosal thickening, pansinusitis, ulcerative lesions in the subglottic area leading to subglottic stenosis and its attendant complications of atelactasis, lobar collapse or obstructive pneumonia are seen in WG [15-17]. Pulmonary involvement in WG ranges from minimal to life threatening comprising of infiltrates or nodules with or without cavitation, and pleural involvement in the form of thickening and effusions [18,19]. Subglottic stenosis in WG needs to be ruled out in all cases as it can lead to respiratory distress with signs and symptoms simulating asthma [15,20]. For the diagnosis of pulmonary vasculitis open lung biopsy is the recommended procedure as transbronchial biopsies provide only small amounts of tissue for examination [19]. However, in view of the high positive test for cytoplasmic antineutrophil cytoplasmic antibodies(c-ANCA) in most of the important group of vasculitides [21] this recommendation has been suggested to be reconsidered. Haji et al observed a perinuclear pattern of ANCA, which can represent antibodies to myeloperoxidase (anti-MPO) and is seen in many inflammatory disorders. Anti-MPO antibodies are found in 20% patients with biopsy proven necrotizing arteritis involving small or medium sized vessels that do not fulfill the criteria for CSS and/or WG [10]. These patients have either asthma without eosinophilia or eosinophilia without asthma suggesting that these patients suffer from a variant of CSS [10].

Hepatitis-B antigenemia which is not infrequently seen in systemic necrotizing vasculitis, is usually not observed in POS. Fauci et al found hepatitis B antigenemia in 6 of the 17 cases which he described as systemic necrotizing vasculitis [13], and 3 of those without antigenemia subsequently were classified as POS whereas none of their 10 subsequently described cases with POS had hepatitis B antigenemia [4].

The clinical course of POS is similar to that in patients without systemic necrotizing vasculitis [4,11,13] having an aggressive disease that ultimately requires therapy with corticosteroids and cyclophosphamide in majority of the cases [6].

No common etiologic link among the disease entities in POS seems to exist, in line with the unclear causation of most of the vasculitic syndromes; the exception being hepatitis B antigenemia (1,6] which can causally be related to a variety of vasculitides from classic polyarteritis nodosa to a cutaneous vasculitic syndrome. Previously hepatitis B associated systemic vasculitis and pulmonary hemorrhage have been reported in a patient [7], which may represent POS. The absence of hepatitis B antigenemia in cases of POS is probably a reflection of the relatively small number of hepatitis B patients developing vasculitis rather than a unique feature of the overlap syndrome [4]. A single antigen may trigger different pathologic responses in different patients as well as elicit varied pathologic responses in an individual patients [4].

The case report by Haji et al in addition to reporting an unusual cause of pulmonary wheezing reemphasizes the recognition of the existence of POS since it is a systemic vasculitic syndrome with the real potential for irreversible organ damage if not treated expeditiously and adequately.

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Fluorouracil Cardiotoxicity

(JK Practitioner 1995; 2:144-5)

5-Fluorouracil (5-FU), a cytostatic antimetabolite is used in various malignancies. Very often high - dose therapy is recommended. Consequently, the toxic effects are also enhanced. Its toxicity against rapidly proliferating tissues like bone marrow., buccal and intestinal mucosae has been well documented [1]. Cardiovascular toxicity is being increasingly reported after the first report by Dent & McColl in 1975 [2]. However, the mechanism/s of the development of these toxic effects remain elusive. In this issue of the *JK Practitioner*, Aziz et al [3] have reported the occurrence of significant bradycardia following 5-F.U. therapy in a young female suffering from adenocarcinoma of colon postulating vagotonia as a mechanism.

There is a varied reported incidence of cardiotoxicity 2.5% to 67% with 5-FU [4,5]. Mir [6] has observed an overall incidence of 83% upon ambulatory electrocardiography. In our experience (unpublished data), some form of cardiovascular abnormality occurs in nearly half the patients receiving 5-F.U but serious toxicity requiring discontinuation of therapy occurs in less than 2%. The fact that cardiotoxicity can manifest in the form of various rhythm disorders [7-9], myocarditis [10], pericarditis [11], angina [2,12], myocardial infarction and cardiogenic shock [13,14] or ST-T changes of cardiac enzyme elevation [5] adds to the postulation that it may be mediated by more than one mechanism. The various mechanisms put forth are (a) autoimmune - mediated injury wherein a complex of 5-FU and cardiac cells stimulates a myocarditic response, (b) direct toxic effects due to the metabolite fluoroacetate, (c) coronary spasm & arteritis, (d) increased thrombogenecity & (e) stunning of myocardium. No single mechanism can explain all the abnormalities observed during 5-FU therapy. Since the effects in a number of patients occur within hours, myocarditis seems an unlikely possibility while coronary spasm or a direct toxic effect or as yet unknown mechanism may be operative in such cases. Coronary spasm is attracting greater attention and prospective studies need to be undertaken to address this postulation although a few studies reported previously have not shown a positive correlation. Aziz et al [3] have postulated yet another mechanism — vagotonia, as their patient showed rapid improvement after atropine administration. It seems unlikely that any of the aforementioned mechanisms is at play. However other tests of autonomic function should be applied before drawing further conclusions. It would still remain elusive whether it was "vagotonia or coronary vasospasm".

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Coronary Heart Disease in Asians Living Abroad

JK Practitioner 1995;2:146-9

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The high incidence of coronary heart disease (CHD) in Asians from the Indian subcontinent living abroad, was first reported by Danaraj et al in 1959 [1]. Several investigators have examined this phenomenon since then, but no coherent view about a clear cut cause has emerged [2-10]. This cause (or causes) should be uniformly applicable to a geographically and culturally diverse group, with varying religious, cultural and culinary habits. Conventional risk factors for CHD do not seem to explain this trend adequately. We examine below, the magnitude of the problem, the role of conventional risk factors, possible effects of insulin resistance and hyperinsulinemia, and the recently recognized influence of the atherogenic lipoprotein, Lipoprotein (a) [Lp(a)] on CHD risk in this group.

While CHD mortality has declined steadily in indigenous western European populations, it has actually increased between 1970-1985, by 8% and 14% respectively in men and women of Asian origin in the United Kingdom [11-12]. Furthermore, between 1979-1983, coronary mortality was higher by 36% in men and 46% in women of Indian origin when compared to the indigenous population in England and Wales [13]. The high relative risk of developing CHD at a younger age than in the native population [13-14], its more widespread nature [8,15] with its implications for mortality and survival [13], and its propensity to affect both sexes [13,16], make this an important health issue. This disturbing trend may be viewed from two standpoints. First, transplanted Asians may have a genetically determined propensity to CHD, and if so, CHD rates and mortality would be similar to that in their places of origin. Unfortunately, comparisons are difficult to make as reliable figures do not exist. There is a difference in the prevalence of major Q waves in men living in urban and rural areas in India [17-18]. The urban prevalence resembled closely CHD rates in British Asians living in Southall [14]. Second, it is also possible that conventional risk factors are qualitatively and quantitatively different in this group of individuals, when compared to the indigenous population. But this is not so for the majority of risk factors. Smoking rates are lower in Asians when compared to the British population (except in Bangladeshis) [19]. Blood pressure levels do not show a significant difference between the two groups either [20-21]. The high variability of dietary constituents in each subgroup within the larger group (e.g. a high fat and energy intake in northern Indians when compared to southern Indians) makes this of doubtful significance too [22]. Physical activity is known to be lower in Asians [23]. The total cholesterol level is also lower in Asians (primarily due to lower high density lipoprotein {HDL} levels) when compared to the white British population. Low density lipoprotein (LDL) levels differ very little [24-25]. However, the implication that cholesterol is unimportant as a risk factor in this population is too simplistic to be acceptable. Studies from Vellore in India, demonstrate a correlation between cholesterol levels and the presence of CHD, although the majority of patients had cholesterol levels deemed to be normal if current 'normal' ranges for Caucasians are accepted [26]. Asian Indians may have a propensity to develop CHD at lower levels of cholesterol when compared to western populations (45% with CHD had levels below 200 milligrams %-approximately 5.2 millimoles/liter) [26].

The prevalence of non-insulin dependent diabetes mellitus (NIDDM) is higher in Asians living in the UK (20% in men and women over the age of 40 years) compared to that in the native European population (about 5%) [27]. This, therefore is a risk factor with a significantly higher prevalence in the Asian community. However the low CHD mortality in Afro-Caribbean people, another ethnic group with a prevalence of diabetes as high as in Asians, would suggest that NIDDM does not

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explain fully the excess risk of CHD in Asians. The hallmark of NIDDM is insulin resistance and compensatory hyperinsulinemia [28]. There is evidence to support a role for hyperinsulinaemia as an independent risk factor for CHD [27,29-30]. It has been suggested that insulin resistance and hyperinsulinaemia are the underlying mechanisms of a syndrome conferring an increased risk of CHD [28]. A constellation of cardiovascular risk factors associated with insulin resistance (high triglycerides, low HDL, hyperinsulinaemia, central obesity and NIDDM), has already been identified in Asian Indian [27,31]. This risk is thought to be mediated by the diverse actions of insulin on cellular and subcellular processes with the potential to produce dyslipidemia, hypertension and a thrombotic tendency [28,32-34]. Asians have an increased prevalence of insulin resistance and compensatory hyperinsulinaemia even in the absence of clinical diabetes mellitus [35-38]. A close correlation between insulin levels in Asians in India and British Asians (who migrated from the same geographical area) suggests that this phenomenon is determined genetically rather than environmentally [38]. Hyperinsulinaemia is closely associated with central obesity [27,37], but is independent of dietary intake in these individuals [39]. There is also a significant inverse relationship between physical activity and insulin levels [38]. The low incidence of CHD in Pima Indians of the United States and in Afro-Caribbeans in the UK, two groups with well characterized insulin resistance and a high incidence of NIDDM, has been cited in objections to the 'insulin resistance' hypothesis. But there are plausible explanations to these exceptions [39].

Recent interest has focused on Lp(a) as an independent risk factor for CHD [40]. Lp(a) is a highly atherogenic subfraction of LDL bearing a close structural homology to plasminogen. Studies from Singapore [41], USA [CADI study - unpublished data], and the UK [42] have shown a consistent elevation of Lp(a) levels in Asian Indians compared to those in the native population. Interestingly, Lp(a) levels are determined genetically and are not affected by drugs, diet etc. [43]. The detrimental effects of Lp(a) are enhanced by high LDL levels or high total cholesterol/HDL ratios (itself, a powerful predictor of CHD in Asians [44]) [45-46]. Lp(a) levels do not change on migration as has been recently shown and may well contribute to the 'genetic' component of the increased CHD risk in Asians [42]. However, of major importance is the fact that Lp(a) reaches adult levels as early as 8 months of age and during infancy may equal levels found in parents affected by CHD [47].

In summary, a high incidence of insulin resistance and hyperinsulinaemia, diabetes mellitus and high levels of Lp(a) in Asians, compounded by low physical activity are some of the factors underlying a high prevalence of CHD in Asians living in the UK and USA. Further studies are necessary to elucidate this important problem. In the meantime prevention of the threatened epidemic of premature CHD in Asians living abroad, should be pursued aggressively. Established risk factors such as smoking,hypertension and hypercholesterolemia should be detected and managed appropriately. "Normal" levels for lipids and thresholds for intervention may need to be revised (downwards) because of the risk for CHD with low levels of cholesterol in this group [48]. Increasing physical activity with improved dietary habits would confer a dual benefit-reduced obesity (improving insulin resistance and its detrimental effects), and a change in the lipid profile to a less atherogenic one (predominantly by increasing HDL). More information is needed before recommendations could be made about genetically controlled Lp(a).

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Catheter - Based Treatment of Coronary Artery Disease Problems, Promises & Future Directions.

(JK Practitioner 1995; 2:149-51)

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Percutaneous transluminal coronary angioplasty (PTCA) remains the standard interventional catheterization approach for the treatment of coronary artery disease (CAD). Recent years have experienced an explosive growth in the use of this technique, probably related to tremendous technological advancement. Despite its wide spread use, significant problems remain for this modality of treatment of CAD. The main concerns are:

1. Chronic total occlusions
2. Diffuse disease
3. Acute occlusion after conventional PTCA.
4. Restenosis

To address these problems, new interventional procedures are being developed and evaluated. There are 4 broad groups of new therapeutic devices and two groups of diagnostic devices.

Therapeutic devices are:

1. Removal devices e.g. atherectomy device
2. Endoprosthesis
3. Lasers
4. Support devices for high risk PTCA

Diagnostic devices are:

1. Intravascular ultrasound 2. Angioscopy.

Removal Devices

Several different approaches to remove or debulk the atherosclerotic lesions have been devised. These devices have been introduced to address the concerns of chronic total occlusions, diffuse disease, abrupt closure after PTCA and restenosis. Various removal devices presently in use are:

(i) ***Atherectomy device (Simpson's Atherocath)***

The device consists of a cylindrical housing, with a balloon on one side which is inflated and acts to position the cutter against the atherosclerotic plaque. The motorized cutter located within the housing rotates at 2500 rpm and shaves off the plaque and removes it from the lumen.

In a multicenter study [1] 1069 lesions in 873 patients underwent atherectomy. LAD lesions were most commonly (64%) treated followed by RCA lesions (26%). The acute success rate was about 85%, mortality rate < 1% and non-fatal MI < 3%. Although initial success rate has been good, long term follow up has shown a substantial incidence of restenosis, ranging from 30% to 50% [2] at 6 months. Due to bulkiness of the device, its use is

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confined to proximal and midsegments of nontortuous coronaries. Due to sharp take off angle, the technique is not well suited for circumflex lesions. Limited success has been achieved in calcific and long segment lesions.

(ii) ***Transluminal extraction catheter (TEC):***
This device has a hollow shaft with surgical blades at tip which rotate at 750 RPM. Cored away plaque is aspirated through the central lumen of the catheter. Stock [3] has reported an acute success rate of 90%; 5% patients required urgent CABG and restenosis rate was about 40% to 50%.

(iii) Rotablator
The concept behind this device has been termed atheroabrasion because it involves rapid rotation (1,50000 to 1,75000 RPM) with an abrasive tip that pulverizes the plaque into particles that are generally smaller than 5*um* which are showered downstream. It has been reported to reduce the mean luminal stenosis diameter from 80% to 45% [4]. Therefore PTCA was often required and 50% patients had restenosis on follow up.

Thus, the currently available removal devices suffer from similar limitations as the conventional PTCA and initial better results are not distinctly advantageous in majority of patients.

Endovascular prostheses (Stents)

The concept behind these devices is to act as a mechanical scaffold, thus either optimizing the earlier results or taking care of dissections caused by conventional PTCA. Initial description was given by Dotter in 1969 [5] when he described use of transluminally placed coilspring endarterial tube grafts in animals. An ideal stent should have:

- rapid, precise & safe placement within the coronary artery
- controlled expansion to a given diameter (2-4mm)
- flexibility to accommodate coronary arteries
- stability to prevent migration once expanded.
- no thrombotic properties
- optimal biocompatibility

Different stents in clinical use are (i) wall stent (ii) Palmaz - Schatz stent (iii) Gianturco - Roubin stent. None of the available endovascular prostheses is ideal. New modifications have been aimed at reducing the need for anticoagulation, improving delivery of the system and preventing or decreasing the rate of restenosis. Polymeric non metallic absorbable stents have been evaluated and have shown promise [6].

Lasers

The ability of laser energy to enlarge stenotic vessel by vaporization of the atherosclerotic plaque rather than by disruption or redistribution of plaque and stretching of vessel wall is appealing. The initial laser devices tested consisted of small single optical fibers coupled to a continuous wave (usually Argon) laser. Foschi & Colleagues [7], reported 76% success and on follow up of 1-13 months, 12 out of 67 patients had reocclusion. Laser thermal-angioplasty and laser balloon-angioplasty tend to have late restenosis in a sizable number of patients. Spectroscopy directed laser-angioplasty has considerable appeal for totally occluded vessels. However complexity, bulk and expense of the procedure are potential disadvantages. Excimer laser angioplasty has been shown to be safe and applicable to long and diffuse lesions [8].

PTCA in high risk patients

Certain groups of patients like unstable ischaemic syndromes, severe LV dysfunction, cardiogenic shock, AMI, single target vessel supplying a large area of viable myocardium, carry a higher risk of complications, mortality and morbidity with PTCA. Several approaches have been developed used either prophylactically before PTCA or to provide hemodynamic stability in the event of any complication. These techniques can be divided into antegrade and retrograde perfusion techniques and cardiac support techniques.

Perfusion techniques

Most commonly used antegrade perfusion device is perfusion catheter that permits passive perfusion of distal coronary bed through side holes in catheter [9]. Alternately, antegrade perfusion can be obtained by coronary hemoperfusion or perfluoro carbon (Fluosol) coronary perfusion. Limitations of such devices are (a) stiffness and large profile of the device which limit flexibility, (b) lack of effectiveness in preventing ischemia of side branch territory and (c) requirement of an adequate perfusion pressure.

Retrograde perfusion can be accomplished by synchronized coronary sinus retroperfusion in which arterial blood is retroperfused through coronary sinus during diastole allowing normal venous drainage to occur in systole. With such a technique, side branch perfusion is maintained and there is no need to cross abruptly-closed segment. It is mainly useful for LAD artery lesions while protection against RCA and circumflex territory ischaemic is unclear. Inability to cannulate coronary sinus may preclude its use.

Support devices

The most commonly used support device is intra-aotric balloon pump (IABP). However it provides only a modest augmentation of cardiac output, and is dependent on stable cardiac rhythm [10]. Another technique, percutaneous cardiopulmonary bypass [11],has gained support because it provides circulatory support in the event of a catastrophic disturbance in cardiac rhythm or cardiac output. However use of this technique involves a high rate of vascular complications (40%), bleeding risk and requirement of a perfusionist. Other less commonly used support devices are Hemopump and partial left heart bypass in which blood is withdrawn from left atrium via transseptal approach and returned via femoral artery.

Conclusion

The problems of interventional cardiology with dilatation remain partly answered. For acute abrupt closure which remains the most dreaded technical complication of PTCA, emergency stenting [12]. laser balloon angioplasty [13] and directional atherectomy [14] have shown promise. Patients with complex angiographic lesions have been helped by directional atherectomy [14]. Degenerated saphenous venous grafts have successfully undergone vacuum extraction atherectomy [3]. Diffuse disease, long lesion and ostial lesions have successfully been tackled by excimer laser angioplasty. A review of the preliminary data available for each of the new devices indicates that the rate of restenosis is quite similar to that of conventional PTCA.[2,8] The relatively high restenosis rate with these devices may reflect a common denominator of initial injury.

A new set of complications has emerged as an aftermath of the new coronary artery procedure. Examples of such complications include (1) coronary artery perforation (2) subacute thrombosis days after stent placement and (3) no reflow phenomenon after rotational angioplasty.

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Duodenal Ulcer Treatment - Progress from pH to HP

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Treatment of duodenal ulcer aims to relieve symptoms, heal the ulcer and reverse the ulcer diathesis. The first two aims have been achieved successfully by reducing acid secretion, both medically and surgically, from the stomach lending support to the dictum 'no acid, no ulcer' [1]. However, acid suppression does not appear to be able to affect the third aim of treatment viz. to reverse the ulcer diathesis because relapse rates are higher after cessation of such treatment [2,3]. Since 1983, *Helicobacter Pylori* (HP) has been incriminated as a major factor in the pathogenesis of ulcer disease, particularly recurrence [4,5]. Undoubtedly the most persuasive argument in supporting the dominant role of HP in duodenal ulcer disease relates to the dramatic decrease of spontaneous relapse after successful eradication of the organism with subsequent healing of the mucosa [6-9]. Thus, although the concept of "no acid", no ulcer" is still valid, yet it should be counterbalanced by "no HP, no ulcer". This has therapeutic implications as was reported by Goodwin [10], in the "leaking roof" concept : it is better to repair your roof (i.e. mucosa) than to stop the rain (i.e. acid). Consequently, duodenal ulcer treatment has progressed in a big way from raising gastric pH to eradication of HP from gastric mucosa.

Duodenal ulcer treatment by raising gastric pH: The formation of duodenal ulcer depends critically on the presence of acid and peptic activity in gastric juice. Schwartz's dictum "no acid - no ulcer" is more accurate if amplified to "no acid and peptic activity - no ulcer" because peptic activity is an indispensable component of the pathogenesis of ulcers and acid without pepsin has little digestive power [11]. The near total dependence of duodenal ulcer on acid and peptic activity is supported by the therapeutic effects of the effective acid suppressing drugs [1]. Acid suppression to raise gastric pH is readily achieved by H_2-receptor antagonists (H_2RAs), proton pump inhibitors (PPIs), synthetic prostaglandin analogues, anticholinergic agents, and new antisecretory agents. The healing rates achieved by acid suppressing drugs correlate with the degree of acid reduction, higher rates being achieved with greater acid reduction (increasing gastric pH) [2]. The anticholinergic agents (pirenzepine and telenzepine) and synthetic prostaglandin analogues (misoprostil) are weak inhibitors of gastric acid secretion [1]. Consequently, these drugs are not used as first line therapy in duodenal ulcer. In contrast, H_2RAs and PPIs have effective acid suppressing activity and are used as first line therapy in duodenal ulcer [1].

H_2 receptor antagonists (H_2RAs)

The currently available H_2RAs include cimetidine, ranitidine, famotidine, nizatidine and roxatidine. These drugs are competitive antagonists for histamine at the H_2 receptor on the basolateral membrane of the parietal cell and decrease basal and nocturnal acid secretion but not meal stimulated acid secretion. In duodenal ulcer patients, gastric nocturnal acid secretion is often inappropriately high. Basal acid secretion occurs mainly at night, and as H_2RAs lower basal acid secretion, their use in the evening should theoretically be beneficial [1]. Approximately 15 years after introduction of H_2RAs for clinical use, most gastroenterologists recommend that a single daily dose be given at bed-time (e.g. ranitidine at 300mg, nizatidine at 300mg, cimetidine at 800, or famotidine at 40mg) [12]. Overall this type of bed-time treatment with an H_2RAs results in approximately 80% of duodenal ulcers being healed after 4 weeks and 95% after 8 weeks of treatment [2,12,13]. When treatment is discontinued after healing of duodenal ulcers with an H_2RA, ulcers recur in 70-90% of patients over the following 12 months [14]. The chronic nature of duodenal ulcer disease and, in particular, the persistent risk of complications have encouraged the adoption of long-term strategies for the management of patients with duode-

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nal ulcer. Long-term therapy can be of value only if it is effective and offers simplicity of dosage and safety. At present the best option for long-term treatment in duodenal ulcer is an H_2RA either as continuous therapy or intermittent symptoms oriented (on demand) therapy with former regimen being superior to the latter [14,15]. All available drugs in this group can be used in a single night-time maintenance dose, invariably arbitrarily set at half the normal ulcer healing dose [15]. On the grounds of simplicity and safety, there is nothing to distinguish between them. The relapse rate is slightly less in patients treated with ranitidine at 150mg at bed-time, compared with those receiving cimetidine at 400mg at bed-time [16]. The relapse rate is even less in patients given ranitidine at 300mg at bed time [17]. Although long-term treatment for an average of 7.5 years keeps most patients free from painful ulcer recurrence and the complications of ulcer disease, it may not lessen the underlying propensity for ulcers to recur once treatment is stopped [18]. It is apparent that treatment with H_2RA should be continued for at least 10 years and possibly indefinitely [19,20]. While considering long-term treatment, it is important to select those patients who need this form of treatment. Many gastroenterologists would accept the following two categories of patients as those most likely to benefit. a) *those who should be on long-term treatment* : elderly and infirm, where surgery may be hazardous due to coexisting disease, post-ulcer surgery recurrent ulceration, patients who bleed from duodenal ulcer without prior symptoms, patients with a perforated ulcer simply oversewn and patients refusing surgery, or who are turned down for surgery; b) *those who may benefit from long-term treatment*: patients with rapid onset, severe symptoms at relapse, patients far from medical aid (sailors, explorers), patients who need NSAIDs or anticoagulants, with a history of duodenal ulceration, and possibly all patients who relapse more than once.

Proton pump inhibitors

The ulcer healing capacity of antisecretory drugs directly correlates with the extent of acid inhibition [2,21,22]. The advent of PPIs that inhibit gastric H^+-K^+-ATPase (final step in acid secretion) has rendered inhibition of gastric acid secretion very effective and convenient [23,24]. It is not surprising therefore that the ulcer healing rates of PPIs surpass those of H_2RAs given at conventional daily doses [25-28]. The currently available PPIs include omeprazole (OME), lansoprazole (LAN) and pantoprazole (PAN). These drugs provide prolonged inhibition of acid secretion regardless of the stimulus and effectively inhibit day-time and nocturnal acid secretion and meal stimulated acid secretion to a significantly greater degree than do the H_2RAs, thus their use in the morning should theoretically be beneficial [23-25]. With the advent of PPIs and their subsequent clinical trials in duodenal ulcer, most authors recommend that a single daily dose be at breakfast (e.g. OME at 20mg, LAN at 30mg or PAN at 40mg) [26]. The world wide 2-week healing rate of duodenal ulcer during treatment with OME at 20mg daily is 66%; the 4-week healing rate is 91%. The early healing rates are an improvement over conventional treatment using an H_2RA, but the minimal period for effective treatment does appear to be 4-weeks. Most patients are symptom free after 2 weeks of treatment, although only two-thirds will have healed their ulcers. The relief of ulcer pain during the first days of treatment with OME is significantly better than with ranitidine during the daytime, but there is no significant difference in symptomatic benefit during night [27]. Similar differences in the healing capacity and symptomatic improvement have been reported in trials comparing ranitidine and other H_2RAs to LAN and PAN [26,28], and this confirms the well known correlation between acid suppression and ulcer healing. PPIs can be of particular benefit in the patients with either resistant duodenal ulcers or whose duodenal ulcer disease is attended by gastroesophageal reflux disease. In patients with resistant duodenal ulcers, OME (40mg in the morning) healed 87% of patients at 4 weeks, compared with H_2RAs (at regular doses) which healed 39%; at 8 weeks, 90% of patients with resistant duodenal ulcers were healed in the OME group compared with 60% of those continuing with regular doses of H_2RAs [29,30].

Clinical development of PPIs has been accompanied by discussions regarding their safety. Whereas adverse effects that are a direct consequence of strong acid inhibition are similar in all these drugs, other side effects may differ. For example, although the known interaction of OME with the hepatic P450 enzyme system is of limited clinical importance, acid suppression with PAN which appears to interact even less with this enzyme system might by preferred [26]. The safety record of OME is excellent, but some recent reports may still indicate true, although rare, side-effects [23]. In these cases, the availability of further PPIs such as PAN may provide a valuable alternative. Unfortunately, there is no "low dose" of PPIs that results in a reliably moderate decrease of intragastric acidity for long-term maintenance treatment. Attempts to induce a mild decrease of acidity using OME (e.g. 10mg daily, 20mg on alternate days, 20mg 3 days per week) failed to yield a regimen that can be relied on for the majority of subjects [31]. OME at 20mg daily is effective for long-term maintenance treatment, but this is achieved by profound inhibition of gastric acid secretion.

As mentioned earlier, maintenance treatment with an H_2RA is not a cure for duodenal ulcer disease; when the long-term treatment is stopped, the tendency for recurrent duodenal ulceration returns [18]. However, HP infection has stimulated great interest in the pathogenesis of duodenal ulcer [5-7,32] and in the 1990s, when doubt no longer exists about the role of HP in curing duodenal ulcer disease [8,9], maintenance treatment with H_2RAs is the second best and it should no longer be considered a primary treatment modality. PPIs in combination with antibiotics effective against HP have shown excellent results in the eradication of this organism [33-35]. HP infection is like any other infection: when it is gone, it is gone. Consequently duodenal ulcer treatment has progressed from increasing gastric pH to eradication of HP.

Duodenal Ulcer treatment by HP eradication:

It is now well established that almost all duodenal ulcer patients, and the vast majority of patients with non-NSAID-related gastric ulcer, are infected with HP [5,6,36]. This HP infection, involving both the gastric antrum and body, affects gastric physiology. In the absence of infection, there exists a tightly regulated negative feedback loop involving somatostatin, gastrin and gastric acidity. HP infection upsets the homeostasis. Somatostatin levels are decreased [37], gastrin levels are high [38] and gastric acid levels are normal or only slightly enhanced which is disproportionate to the stimulus [5]. The consequent chronic active inflammation weakens the defensive capacity of the mucosa against acid-peptic attack, ultimately resulting in ulcer formation [6,7]. Despite its ubiquity and persistence, only a small proportion of HP-infected persons develop clinical consequences, such as peptic ulceration or neoplasia [5]. At most, those developing these illnesses represent less than 10-20% of all infected persons. Understanding the reasons for this important dichotomy represents the major current challenge in HP research. Factors [5] that could influence this phenomenon are I) virulence differences of HP strains II) genetic differences among infected hosts III) exposures to other environmental agents IV) age at exposure to HP.

There is a dramatic decrease of spontaneous ulcer relapse after successful eradication of the organism with subsequent healing of the mucosa [6-9]. This dramatic reduction in ulcer relapse led a National Institute of Health consensus conference to advise anti-HP treatment as first- line therapy for all HP associated duodenal ulcer and gastric ulcer disease [39]. These impressive

Helicobacter Pylori

results persist as long as no recurrence of the infection occurs. If a higher re-infection rate is found, one should seriously question whether the organism,was truly eradicated, or whether one is dealing with recrudescence of the infection after only temporary suppression [40]. For these reasons, an attempt at eradication should be the first-line approach for all patients with HP associated duodenal ulcer disease [6,11].

Antibiotic regimens used for HP eradication.

Antibiotic regimens used for HP eradication are either conventional bismuth-based triple therapy or with addition of a PPI (OME/LAN) to the conventional antibiotics effective against HP [35]. The addition of a PP1 is based on the observation that "anacidity enhanced antibiotic treatment." The possible mode of action of PP1s are [6] : 1) competition from other overgrowing micro-organisms, 2) autotoxicity from unbuffered ammonia, 3) direct antimicrobial effect, 4) synergy with other antimicrobials,5) increased topical concentration of antimicrobial, 6) optimizing antimicrobial efficacy through enhanced pH, 7) strong anti-urease effect, 8) effect on bacterial metabolism (urease production? ATPase activity?) and migration, 9) effect on host immunity (immunoglobulin breakdown; improved polymorphonuclear leukocyte function) and, 10) combinations of the above.

It becomes almost impossible to summarize the many regimens which have been used in HP eradication therapy. For practical purposes, and to simplify the issue, one may distinguish the following treatment categories.

1)Bismuth based triple therapy:
    [Bismuth + tetracycline or amoxicillin or spiramycin + imidazole (metronidazole or tinidazole)].
2)Bismuth based dual therapy:
    [Bismuth plus one antibiotic (metronidazole or amoxicillin or ofloxacin)].
3)OME based quadruple therapy:
    [OME+bismuth based triple therapy].
4)OME based triple therapy:
    [OME+two antibiotics(amoxicillin+clarithromycin)or(amoxicillin+metronidazole)or (clarithromycin+imidazole)]
5)OME based dual therapy:
    [OME+oneantibiotic(amoxicillinor clarithromycin)].

The optimum regimen for the eradication of HP remains unclear. This is illustrated by the large number of treatment regimens and protocols reported in the literature. The best and most consistent results are obtained with bismuth based traditional triple therapy [6]. Equally effective is combination therapy of a PP1 (OME) with two antibiotics [42] or traditional bismuth based triple therapy [43]. However, these combination therapy regimens are fraught with many disadvantages: (a) resistance to the antibiotics, especially metronidazole and tinidazole, particularly with prior therapy with these agents [44,45], (b) a high side-effect profile [46,47] and, (c) poor compliance due to the large number of tablets required to be taken daily for a minimum period of 2 weeks [46,47]. For these reasons gastroenterologists and others have been motivated to look for alternatives.

The observation that OME rapidly relieves symptoms, suppresses HP directly, and increases the effectiveness of several antibiotics and has led to its wide spread use in various eradication regimens [27,40,48-50]. Dual therapy, comprising OME and amoxicillin, has been advocated as having the advantage of simplicity, few side effects and good compliance when compared to bismuth based triple therapy [34,43]. However the geographical scatter of results with such dual therapy is surprisingly high, which is unfortunate. Whilst some authors have consistently found eradication rates over 60% [48,50] others have reported eradication rates of 25% to 60% only [33,35,49]. Moreover, this dual therapy alone appears to be less effective than dual therapy in combination with metronidazole which is considered as an important component of HP eradication regimens [35]. With this perception of literature, dual therapy (PPI plus one antibiotic) will ultimately prove insufficiently effective in the eradication of HP until more effective dual therapy, possibly with different antibiotics is demonstrated. Consequently most regimens will continue to require two antibiotics. In accordance with the conclusion of the recent consensus Development conference on HP at the National Institute of Health (NIH), Bethesda, MD, USA (February 1994) all ulcer patients should receive treatment with a combination of antibiotics. The optimum length of antibiotic therapy in the eradication of HP is unclear. Currently most interest is focussed on 1-week therapy of two antibiotics (amoxicillin plus metronidazole) in combination with a PP1 (OME), with excellent results [51,52].

The mainstay of anti-HP therapy is not only to prescribe an efficient drug combination, but also to prepare and motivate the patient into taking the medication as prescribed. We might have to invest sometime in doing so, but curing this infection is rewarding because it greatly improves the quality of life of our ulcer patients [53-55]. However, many questions still need to be answered. What are the best drugs and the best combination? what are the most cost-effective doses required for both ulcer healing and HP eradication? How safe are these combinations? What is the risk of ulcer rebleeding after eradication of HP? Many of these questions may be answered shortly but it is now clear that duodenal ulcer is an infectious disease and like any other infection: when it is gone, it is gone.

To conclude, treating HP infection is still an art and we learn more month by month. In view of the recently published trials, we are currently recommending that all HP positive duodenal ulcer patients should be treated with a combination of OME (20 mg BD), amoxicillin (500 mg TID) and metronidazole (400 mg TID) for 1-week, to achieve the third aim of duodenal ulcer treatment (to reverse the ulcer diathesis), which is not possible with the conventional acid suppressing drugs [18]. Moreover after successful eradication of HP no maintenance treatment is required in contrast to duodenal ulcer treatment by acid suppressing drugs [55]. Thus duodenal ulcer treatment has progressed in a big way from control of gastric pH to eradication of HP.

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Original Article

Maternal Epidemiological Correlates in Birth Asphyxia

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Abstract: Of 1651 babies born during the study period of December 1992 to December 1993, 69 (4.2%) had birth asphyxia; being mild in 32, moderate in 19 and severe in 18 cases. Various maternal correlates identified to have an association with birth asphyxia included age less than 25 years (p.<001), multiparity (p<.001), spacing of less than 18 months (p<.001), toxemia of pregnancy (p<.001), anemia (p<.001) and diabetes (p<.001). Socioeconomic maternal correlates included non-working mothers, illiteracy and a low family income. *JK Practitioner 1995; 2:159-60.*

Key words: Birth Asphyxia, Maternal correlates, Apgascore
Abbreviations: AS = Apgar score

Introduction

Birth Asphyxia is one of the commonest and leading medical emergencies in newborn. It accounts for 5.3% perinatal deaths in developing countries as compared to 1.5 -2% in developed ones. Its incidence is 5-10% in India as compared to 1.5% in developed countries [1-4]. Toxemia of pregnancy, maternal diabetes and sub-optimal obstetric care have been found as strong correlating factors for birth asphyxia [5-7]. Our study was aimed to find various maternal correlates which lead to birth asphyxia in newborn.

Material and Methods

The study was conducted at Maternal and Child unit, department of Community Medicine and the department of Neonatology, Institute of Medical Sciences, Srinagar from December 1992 to December, 1993. Complete antenatal history was taken. Various maternal correlates which were studied included maternal age, parity spacing, antenatal care, type of delivery, toxemia of pregnancy, anemia, diabetes, occupation of mother, educational status of mother and monthly income of family.

Three hundred and sixty nine mothers were selected from a total of 1651 mothers to study various correlates, including all the 69 mothers whose babies had birth asphyxia, and 300 controls; sampling being done by census based technique. Each mother was monitored during delivery. Birth asphyxia was labeled according to the Virginia Apgar Scoring system [8]. Birth asphyxia cases were divided into three grades according to their Apgar scores (AS), mild (AS 7-8), moderate (AS 4-6) and severe (AS 0-3). 300 newborns with AS of 8 or more were kept as controls.

Results

Out of 1651 babies born during the study period, 69 had birth asphyxia. 32(1.94%) had mild birth asphyxia, 19(1.15%) had moderate asphyxia and 18(1.09%) had severe birth asphyxia.

Various maternal correlates in birth asphyxia patients are given in table 1.

Maternal correlates	Number	No. with birth asphyxia (%)
1.Maternal age		
<25 yrs	47	20 (42.55)
25-30 yrs	234	31 (13.25)
>30 yrs	88	18 (20.45)
2.Parity Primiparous	57	22 (38.60)
Multiparous	312	47 (15.05)
3.Spacing inmothers		
<18 months	93	37 (39.78)
18-24 months	162	19 (11.73)
>24 months	114	13 (11.40)
4. Antenatal care		
No antenatal care	22	12 (54.55)
Partial or incomplete antenatal care	231	43 (18.61)
Complete antenatal care	116	14 (12.07)

Table 1. Maternal correlates of birth asphyxia babies.

Birth asphyxia was observed in greater frequency in mothers of <25 years of age compared to those with >25 years (p<.001), and in multiparous moth-

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ers compared to primis (p<.001). Mothers having done spacing for more than 18 months had less prevalence of birth asphyxia as compared to those mothers who did spacing of less than 18 months (p < .001).

Toxemia of pregnancy, anemia and diabetes in mothers were identified as the medical maternal correlates associated with birth asphyxia. 58.62% babies of mothers with toxemia, 48.61% babies of anemia mothers and 75% babies born to diabetic mothers suffered from birth asphyxia. All these values were statistically highly significant (p<.001). The various maternal medical correlates are shown in table 2. Other correlates such as socio-economic status, educational status of mother and monthly income of the family are shown in table 3. House wives had higher chance of birth asphyxia in babies, when compared to working ladies (p < .001). Illiterate mothers had higher chances of giving birth to asphyxiated babies as compared to literate mothers (p <.001). Mothers from low socioeconomic groups had higher chances of birth asphyxia in babies as compared to higher socioeconomic group (p < .001) [Table-3].

Maternal medical correlate	No. of new-born	Birth asphyxia N(%)
Toxemia of pregnancy		
Yes	29	17 (58.62)
No	340	52 (15.29)
Maternal anemia		
Yes	72	35 (48.61)
No	297	34 (11.45)
Maternal diabetes		
Yes	08	06 (75)
No	361	63 (17.45)

Table 2. Maternal medical correlates in birth asphyxia.

Socioeconomic correlates	No. of new-borns	Birth asphyxia (%)
Occupation of mother		
House wife	226	58 (25.66)
Employed	143	11 (07.69)
Educational status		
Illiterate	168	52 (30.95)
Literate	201	17 (08.46)
Monthly family income		
<Rs.1000	118	34 (28.81)
Rs.1000-1500	149	23 (15.44)
> Rs.1500	102	12 (11.76)

Table 3. Socioeconomic correlates of birth asphyxia.

Discussion

Birth asphyxia is one of the leading causes of potentially preventable perinatal mortality. Overall prevalence of birth asphyxia was 4.18% in our study as compared to 5-10% at the national level [13]. A lower prevalence in Kashmir as compared to rest of India may be due to better medical care in this state. The prevalence of severe birth asphyxia was 1.09% in our study as compared to 3.2% in another Indian study [11]. Our study showed that birth spacing of 24 months and proper antenatal care decrease the incidence of birth asphyxia significantly.

Toxemia of pregnancy showed a highly significant correlation with birth asphyxia with of 58.62% as compared to 15.29% in non-toxemic mothers. Toxemia of pregnancy is associated with intrauterine growth retardation and prematurity which can lead to birth asphyxia. Similar findings were observed by others [5,11]. Diabetes in the mother is associated with birth asphyxia in the newborn as the placental perfusion is reduced upto 50% in diabetes [12]. Maternal anemia also a bore highly significant association with birth asphyxia. If maternal anemia, diabetes and toxemia are taken care of in pregnancy, the incidence of birth asphyxia can be decreased markedly.

Various socioeconomic variables that were observed to affect the frequency of birth asphyxia included maternal occupation, education and monthly family income. No attempt has been made till date to explore the association of socioeconomic correlates with birth asphyxia as it is generally presumed that birth asphyxia is purely related to obstetric, medical or neonatal phenomena. Epidemiologically speaking factors could occur in a tier system which could act as a stimulus from the grass root level to the intervening one and the intervening factors could precipitate the final causative factor.

Visualizing the interplaying role of the factors studied in the present series it is felt that most of the factors are preventable in nature. The factors could be controlled or eliminated by means of extensive health education and antenatal care through primary health care approach.

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Original Article

Epithelial Dysplasia in Cancer Stomach

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Abstract: Detailed microscopic study of mucosal changes was conducted in 100 patients of gastric carcinoma attending Gastroenterology department of Medicine, SMHS Hospital, Srinagar, Kashmir. Upon histological typing, 57 were intestinal type, 43 were diffuse type of gastric carcinomas. Epithelial dysplasia was observed in 19 cases (19%) which was present in the gastric mucosa surrounding both intestinal type as well as diffuse type of gastric carcinomas. *JK Practitioner* 1995; 2: 160-62.

Key words: Gastric carcinoma, Dysplasia, Precancerous conditions

Introduction

Cancer of the stomach is one of the commonest and most serious forms of malignant disease in native population of Kashmir. Durrani et al, in 1988 [1], in a study of 850 cases among Kashmiri native population over a period of five years reported gastric carcinoma as the most frequent neoplasm of gastrointestinal tract. Mattoo et al (1974) have reported a prevalence of (28.%) among all malignancies in Kashmir [2]. Such a prevalence is comparable to high prevalent areas of the world, such as China, Japan, Iceland etc. The etiological possibilities for gastric carcinoma that have attracted attention include genetic, dietary, racial and geographical theories, as well as mucosal changes in the gastric musoca. The mucosal change of epithelial dysplasia as a risk factor and its relation with gastric carcinoma is the concern of this paper.

Material and Methods

The study included 100 cases who were subjected to endoscopic examination and biopsies taken were subsequently confirmed as cases of gastric carcinoma by light microscopy. Endoscopic biopsies were taken from the mucosa immediately surrounding the growth and also at a distance of 1cm from the growth. The tissues were fixed in 10% neutral formalin and were processed in automatic tissue processor. Paraffin blocks were made and 3-4 u thick sections were cut and stained by hematoxylin and eosin stain. Detailed histological study of the slides was done and gastric carcinomas were classified as per Lauren (1965) [3], and as also quoted by Robin, Cotran and Kumar (1984) [4] into intestinal type of gastric carcinomas and diffuse type of gastric carcinomas. Mucosal changes were studied. Epithelial

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dysplastic changes were noted wherever present and were classified as per Grundmann (1975) [5], as mild, moderate or severe.

Results

In the 100 cases of gastric carcinomas studied, 78 tumors were located in the distal portion of stomach whereas 22 were in the proximal portion. The mucosal changes observed included atrophic gastritis, intestinal metaplasia and epithelial dysplasia. Nineteen cases (19%) demonstrated epithelial dysplasia in the mucosa immediately surrounding the tumor and also in he mucosa distant from the tumor. The histological and cytological features of epithelial dysplasia seen were disorganized mucosal architecture, abnormal differentiation and cellular atypia. Moderate type of epithelial dysplasia was seen alongside intestinal metaplasia in 3 cases (16.7%) of intestinal type of gastric carcinoma whereas severe dysplasia was observed in 16 cases (83.2%) of the 19 cases of diffuse type of gastric carcinoma. The dysplastic changes were in the age group of 51-60 years and males outnumbered the females.

Discussion

19% of tumors had associated epithelial dysplasia in the present study. Meister et al (1979) [6] have observed moderate epithelial dysplasia to the extent of 90% and severe dysplasia to the extent of 40% in 10 cases of early gastric carcinoma [6]. Moreover, severe dysplasia was seen in the mucosa immediately surrounding the cancerous mucosal lesions. Our observations and those of the authors are comparable.

Grundmann (1976) [5] has reported the significance of mucosal lesions referred to as dysplasias of different grades as cancer precursors and these dysplasias comprise of alterations of epithelium with various degrees of differentiation and proliferation [5]. Morson et al [7,8], have emphasized the importance of epithelial dysplasia in the stomach as a marker for increased cancer risk. The histological and cytological features are comparable with that of the authors.

Low grade dysplasia in the stomach does not usually progress to severe dysplasia or cancer, whereas severe dysplasia does progress, according to some observers [9]. Therefore, frequent follow up is warranted in these cases. Bioptic diagnosis of severe dysplasia should always point towards intensive search for cancer in the stomach. In cases of severe dysplasia in association with a polyp, ulcer, or hypertrophy of gastric mucosa, resection should be performed.

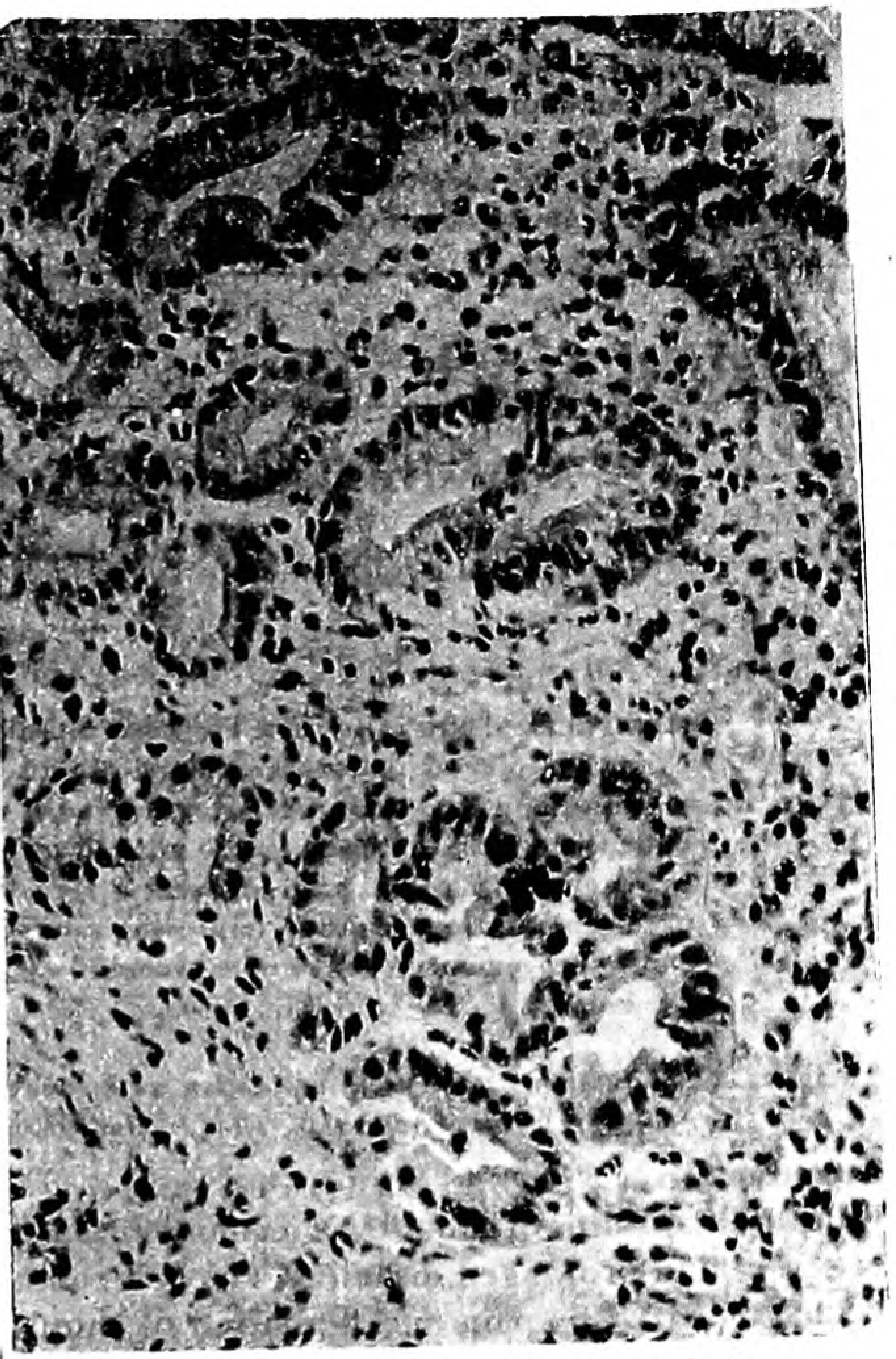

Fig.1. Photomicrograph showing epithelial dysplasia

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Original Article

Adhesive Capsulitis - Treatment with Oral Steroids

Bashir A Buth

Abstract: Sixty patients of adhesive capsulitis shoulder were studied. The patients were randomly placed in two treatment groups. One group received short course of oral prednisolone, and the other group served as controls receiving no specific treatment. The patients in both the groups were advised to perform pendular exercises at home. The improvement in night pain showed a significant difference in two groups, with the prednisolone treated group improving rapidly. The recovery in pain at rest, on movement and improvement in range of movement was not significant. *JK Practitioner 1995; 2: 162-4.*

Key words: Adhesive capsulitis, steroids

Abbreviations: MUA = manipulation under anesthesia, FLX = Flexion, EXT = Extension, ABD = Abduction, ADD = Adduction, IR = Internal Rotation, ER = External Rotation.

Introduction

Adhesive Capsulitis of shoulder is a common cause of severe and prolonged disability. The syndrome is characterized by pain and limitation of shoulder movement in absence of any recognized intrinsic abnormality. It was first described by Putman in 1882 [1] and latter by Codman [2]. The initial presentation is the pain, which is generalized and referred to upper arm, the back and the neck. The pain is more severe in early stages and disturbs sleep. The limitation often persists even after prolonged follow up [3,4].

The pathogenesis of adhesive capsulitis is uncertain. De Palma reported that any condition that hindered scapulohumeral motion, caused muscular inactivity and predisposes the patient to adhesive capsulitis [5]. Neviaser found capsular adhesions underlying humeral head upon surgical exploration for adhesive capsulitis [6]. Macnab [7] suggested that partial interruption in blood supply to a tendon can cause degeneration of tendon collagen followed by a type IV auto immune reaction. An association between adhesive capsulitis and HLA B27 antigen was subsequently reported [8].

It is unclear whether the contracture of the shoulder capsule is a passive process related to lack of motion or an active process associated with capsular inflammation. Wide range of therapeutic regimens have been advocated for treatment of adhesive capsulitis. These include local steroid injections [9,10], manipulation under anesthesia [11,12], physiotherapy [13,14], traction [15], radiotherapy [16] and stellate ganglion blocks [17]. All these therapeutic measures have no long term advantage. Blockey et al [18] compared oral steroids to placebo in 31 cases, and reported a significant improvement in pain but not in range of movement. Kessel et al [19] found manipulation under anesthesia (MUA) and oral steroids more effective than MUA alone. Lloyd-Roberts and French [9] compared steroid injections and MUA to oral prednisolone and found the former regimen better. Binder et al [20] in a controlled study reported improvement in pain but not in range of motion. Non steroidal anti-inflammatory drugs are widely used in the treatment of adhesive capsulitis but are of doubtful value [21-23].

The present study was aimed to ascertain the possible beneficial effects of a short course of oral

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prednisolone therapy in patients of adhesive capsulitis.

Material and Methods:

Sixty (60) patients who complained of pain and stiffness of the shoulder were studied. The patients were selected according to the following criteria [24].

1.Spontaneous onset of pain localized to shoulder region, pain increasing in severity and usually worse at night.

2.Limitation of all shoulder movements by at least 50%.

3.No clinical and radiological identification of a lesion of shoulder.

All these patients had restriction of active and passive movements of shoulder with pain and sleep disturbances of at least 1 month duration.

The patients with history of generalized arthritis, peptic ulcer, diabetes, hemiplegias, serious infections, cervical radiculopathy and any contraindications to systemic steroids were excluded from the study.

There were 34 male and 26 female patients, the age range was 45-70 (mean 55.4). The duration of presentation varied from 1-12 (mean 6.0) months. 41 patients had involvement of non dominant shoulder, and 19 of the dominant shoulder. The onset of symptoms was spontaneous in all the patients, 10 patients had history of mild trauma.

Pain radiographs of shoulder and hemograms in all patients were normal. HLA B27 antigen was done only on 6 patients and was reported absent. The clinical assessment was done before the treatment and at the end of 6 weeks, and thrice in next 6 months at 2 months intervals.

Pain was recorded as pain at night, pain on movement and pain at rest during the day.

The range of passive shoulder movement was recorded by a goniometer in six movement parameters of Flexion (FLX), Extension (EXT), Abduction (ABD), Adduction (ADD), Internal rotation (IR) and External rotation (ER). The variations between the movement range at the initial visit, subsequent visits and at the end of 6 months were recorded.

The patients were randomly placed in one of the two treatment groups.

A.*__Treatment group (30 patients)__* Prednisolone 10 mg. was given to these patients as a single morning dose for a period of 4 weeks. The dose was then reduced to 5mg daily for another 2 weeks, and stopped thereafter.

B.*__Control group (30 patients)__* These patients were allowed to take non salicylate analgesic agents and diazepam if the pain was not tolerated and had sleep disturbed.

The patients in both the study groups were advised to perform pendular exercise for 2-3 minutes every hour at home.

Results:

All the patients before entering the study had received treatments with various drugs mostly NSAIDS, but only nine (15%) reported some improvement in pain. The patients had not received any advice on physiotherapy or general management of shoulder. Only 10 patients had been advised to gently move the shoulder and others to rest the shoulder.

	Initial visit		At 6 months	
	Mean	Range	Mean	Range
Movement				
FLX	78°	50-90°	95°	80-110°
EXT	26°	20-30°	38°	30-50°
ABD	78°	60-90°	96°	80-110°
ADD	25°	20-30°	37°	30-45°
IR	31°	25-45°	46°	25-65°
ER	31°	25-45°	48°	30-65°

Table-1. A comparison of range of movement in patients at initial visit and at 6 months of followup.

At presentation 36 patients had pain at night and had disturbed sleep, 10 patients had pain at rest and 14 patients complained of pain on movement. On examination at the first visit the mean and range of each movement parameter was as follows: FLX 78° (50-90°); EXT 26° (20-30°); ABD 78° (60-90°) ADD 25° (20-30°) IR 31° (25-45°); ER 31° (25-45°). The recovery in range of movement was recorded in two treatment groups on subsequent visits and at the end of follow up (Table-1). There was no significant improvement in treated group in recovery of individual movement parameter. Arm dominance did not influence the outcome (Table-2).

The two groups showed initial recovery in pain. The improvement in pain at night and pain at rest, showed severe pain when prednisolone was withdrawn, which subsided spontaneously.

All patients showed improvement in pain during the course of study. 10 still had pain at the end of

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6 months. A comparison of the range of movement in two groups was insignificant.

	Oral Steroid group (30 patients)		Control group (30 patients)	
	Initial visit	6 months	Initial visit	6months
Movement				
FLX	78°	95°	79°	98°
EXT	25°	37°	26°	40°
ABD	76°	93°	80°	99°
ADD	25°	37°	24°	36°
IR	29°	47°	32°	45°
ER	31°	46°	31°	46°

Table-2. Comparison of mean range of movement in the two groups at 6 months follow up

Discussion:

The short course of oral prednisolone in some patients of adhesive capsulitis with intense pain and sleep disturbances is useful. This was not associated with any serious side effects. Severe pain recurred only in 4 patients when prednisolone was stopped, NSAID were found to be of little value. 10 patients only had been advised to gently move the shoulder. Both the groups were advised home pendular exercises, A marginal improvement in patients in No treatment group suggested that pendular exercise may be important in initiating recovery.

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Original Article

Late Presentation in Cancer: Who is Responsible ?

MA Teli [*], *SU Kuchay* [**], *MY Kharadi* [***], *MM Lone* [****]

Abstract: Cancer is becoming a major health problem in many developing countries including India. Unfortunately, 80-90 % of our cancer patients present in late stage of their disease when it has either disseminated or is locally advanced to such an extent that the only therapeutic option is use of a palliative approach. From 1993-95 data of such 250 patients with various late stage cancers was analyzed in our department to unearth the cause for this late presentation. It was surprising to observe that contrary to our belief, medical community is responsible for this time delay to a great extent. All this calls for a reappraisal of our attitude to cancer problem. *JK Practitioner 1995; 2: 165-8.*

Key words: Cancer, Late presentation, Diagnosis

Introduction

The global incidence of various cancers is on rise and in fact, cancer has become a major health problem in many developing countries. Though, regarded as a dreaded and mysterious disease in past with no known cause and no known cure, cancer is no longer an incurable disease today. Last two decades have witnessed tremendous achievements in medicine and biomedical technology which have revolutionized the management of cancer. Today, it has become possible to cure 1/3rd of cancers, prevent 1/3rd and palliate the rest of them. However, a timely and quick therapeutic intervention plays a very crucial role in management of these diseases. Because, outcome and time factor go hand in hand in cancer management and any delays in diagnosis and/or treatment result in disastrous consequences.

Statistically, the outcome of cancer patients in developed countries is excellent compared to the situation in developing countries. Unfortunately, 80-90 % of our patients present with late stage disease when the best therapeutic option for majority of these patients is use of a palliative approach [1]. Traditionally, we the medical community have been blaming patient ignorance for this late presentation. However, late presentation is not unusual even in literate and well off families. In this study our aim was to explore the causes of this late presentation and to find who really is responsible.

Material and Methods

The study was conducted on 250 patients of late stage cancers (stage III & IV) of various histopathologies and anatomical sites. The break-up of these patients is shown in table-1.

Esophagus	75	Lung	31
Skin cancer	39	Head & neck	25
Metastatic lymph nodes	19	Soft tissue sarcoma	15
Breast cancer	7	Gynecological	15
GIT. (excluding esophagus)	12	Genitourinary	8

Sex Male:female: 3:1
Mean age 56.23±10.28 years
Range (17 years to 69 years)

Table-1: Break-up of the various malignancies.

A proforma (see proforma) was given to the patients to be filled in the department. in isolation or in presence of a literate attendant in illiterate patients. The process was supervised and guided

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by one of the doctors of the department. The consultation dates were cross checked from various sources like private prescriptions, Investigation forms etc. Cases without documentary proofs were not included in the study.

PROFORMA

NAME :
R.T.NO.:
Malignancy
Stage:

- When Patient was absolutely alright (Day/month/Year) ________
- Months/Years back symptoms/ symptoms started ________
- Ignored for many days/months/years ________
- Consultation with Hakim (if any) for how many days/months/years ________
- Consultation of any peer/Fakir/Hajam for how much period. (Days/months/ years ________
- Consultation of a compounder (specify period in days/months/years) ________
- Consultation of a dispensary doctor (specify period in months/years) ________
- Consultation with a specialist in General Medicine/Gen.Surgery/ Obstetrics/Gynae/ENT/Ophthalmology ________ (Specify period in days/months/years)
- Consulted SMHS Hospital for how much period. (Days/months/years) ________
- Attended Radiotherapy Department Specify day/month/year) ________

Results

The results of the study are shown in table-2. Average time delay was 6.5. months. The time delay attributable to patient ignorance was 2-8 weeks compared to 19 weeks delayed by the medical community ($p < 0.5$). It was observed that there is a general tendency among patients to ignore their symptoms for 2-5 weeks with a mean duration of 3±0.2 weeks. Consultation was sought earlier in symptoms of bleeding and was delayed for painless swellings & non bleeding ulcers.

There are quacks in society who claim to have healing touch and consequently treat each and every patient who approaches them for any symptom. This tendency of consultation was observed in illiterate, rural and patients of low socioeconomic status. Overall, consultation of these quacks was sought by 65% of our patients and led to a delay of another 5-8 weeks, With a mean delay 6.0±1.3 weeks.

General practitioners were responsible for managing the patient conservatively for another 2-4 weeks and the specialists for another 7-9 weeks. Unfortunately, the investigations asked to be carried out by these personnel, especially by the general practitioners, were either ignored totally (63% patients in G.P consultation Vs 17% in specialist consultation) or were carried out after some delay (7% in G.P consultation Vs 29% in specialist consultation). 3% patients had not carried out the investigations asked either by the G.P or the specialist.

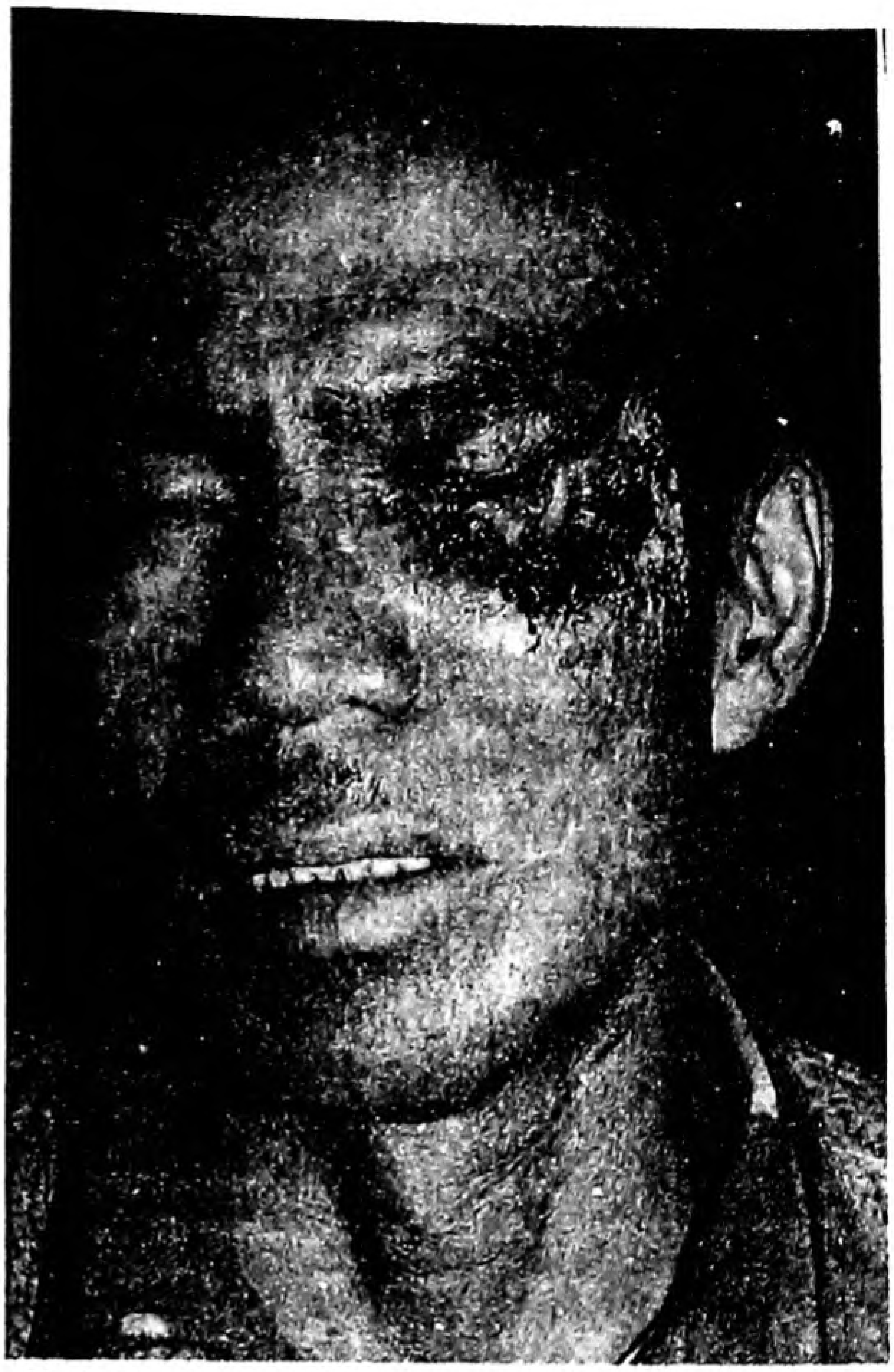

Fig.1. Basal cell carcinoma of lower lid eroding the orbit (symptoms started 6 months back) - T4, N2,M0

A further delay of 1-9 weeks was seen added to the already delay in consultation in district/referral hospitals and in carrying the various investigations. A trend of in-coordination was observed amongst various specialities.

The sequence of consultations shown in Table-1 (we termed it "regular sequence") was observed in 85% patients. However, irregular consultation sequence was observed in 15% patients i.e. some initially going to G.P first. and then to paramedics or to hospitals first and then to private specialists and so on. It was also observed that time delay was more in younger (5.9 vs. 5.1 months) and female

(5.8 vs. 5.2 months) patients. However, neither of these was statistically significant.

Discussion

While, there may be a reasonable disagreement amongst oncologists regarding the natural history of cancer, all of them agree on the point that "earlier a cancer is detected and treated; better the results of therapy." And as the prognosis in these diseases is related to time factor, a timely diagnosis and treatment plays a very crucial role in management of these diseases, because first chance to cure is often the only chance to cure these diseases. Our results in this regard are not encouraging when we compare them with the results in developed countries. The main reason for our dismal results is that 80-90 % of our cancer patients present with locally advanced and/or disseminated diseases when the only therapeutic option available is the use of a palliative treatment for relieving the distressing symptom/symptoms.

S.No.	Cause	Time delay (wks) Range (Mean±SD)
i.	Patient ignorance	02-05 (3.0±0.2)
ii.	With quacks,peer,fakir, hakim,paramedics	05-08 (6.0±1.3)
iii.	General practitioners	02-04 (2.3±1.5)
iv.	Specialists (private)	07-09 (8.2±1.4)
v.	District:hospitals/referral hospitals	05-09 (6.8±1.3)
vi.	Investigation procedures	01-04 (2.0±0.5)

Table-2. Time delay in treatment and its causes. Average time wasted from start of symptoms to initiation of specific treatment - 06.50 months

Both patient & medical community are responsible for this scene. However, contrary to our notion, it is the medical community who are contributing a lion's share in this regard (Time factor approximately 2.5 times greater than wasted by patient factor). Some of our medical colleagues have developed a rather nihilistic attitude towards cancer by giving in all hopes once a diagnosis of cancer is established in a patient. How unrealistic! Today, with many advances in various sub-spe

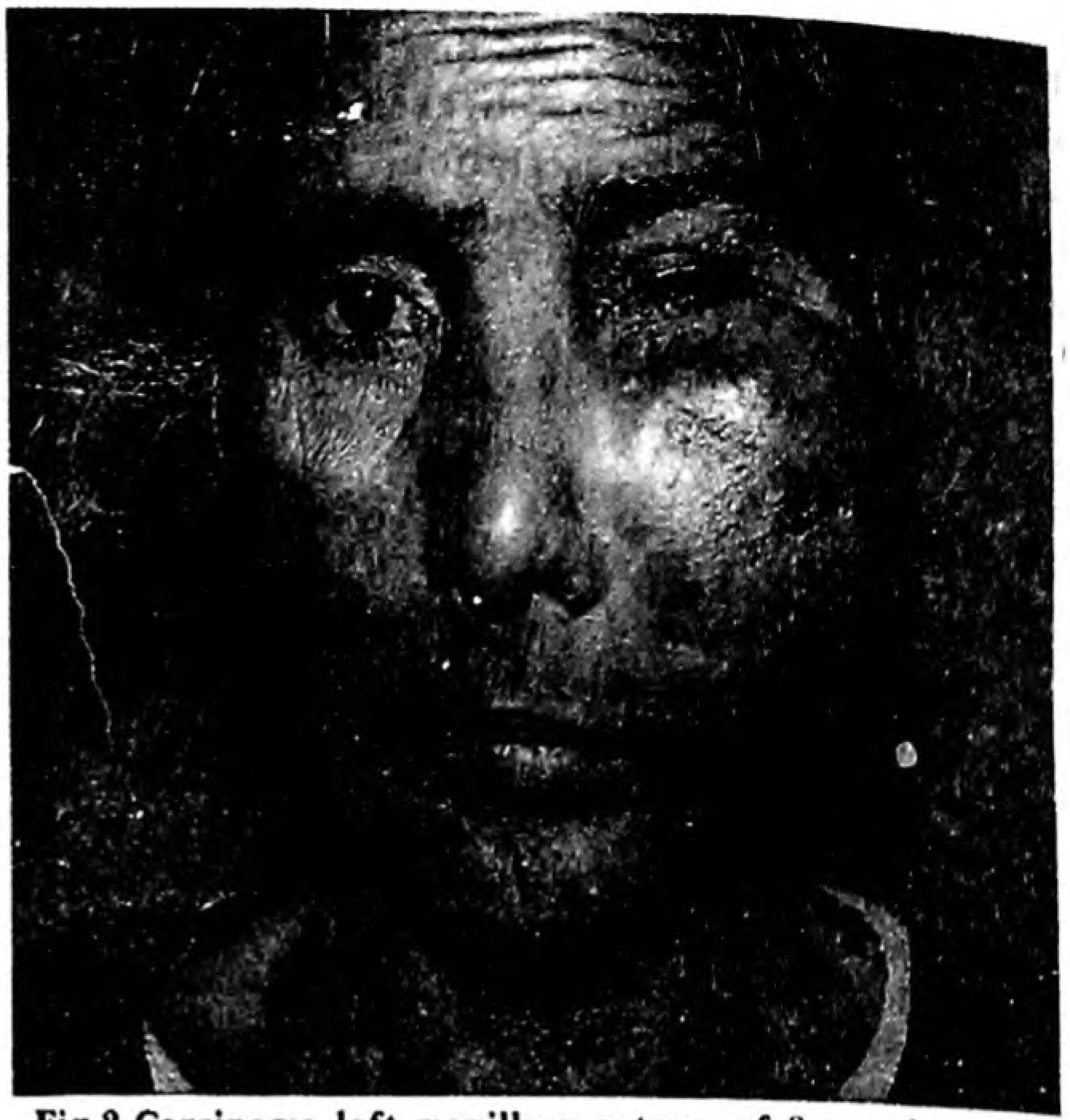

Fig.2.Carcinoma left maxillary antrum of 8 months duration, about to fungate (T4, N1, M0).

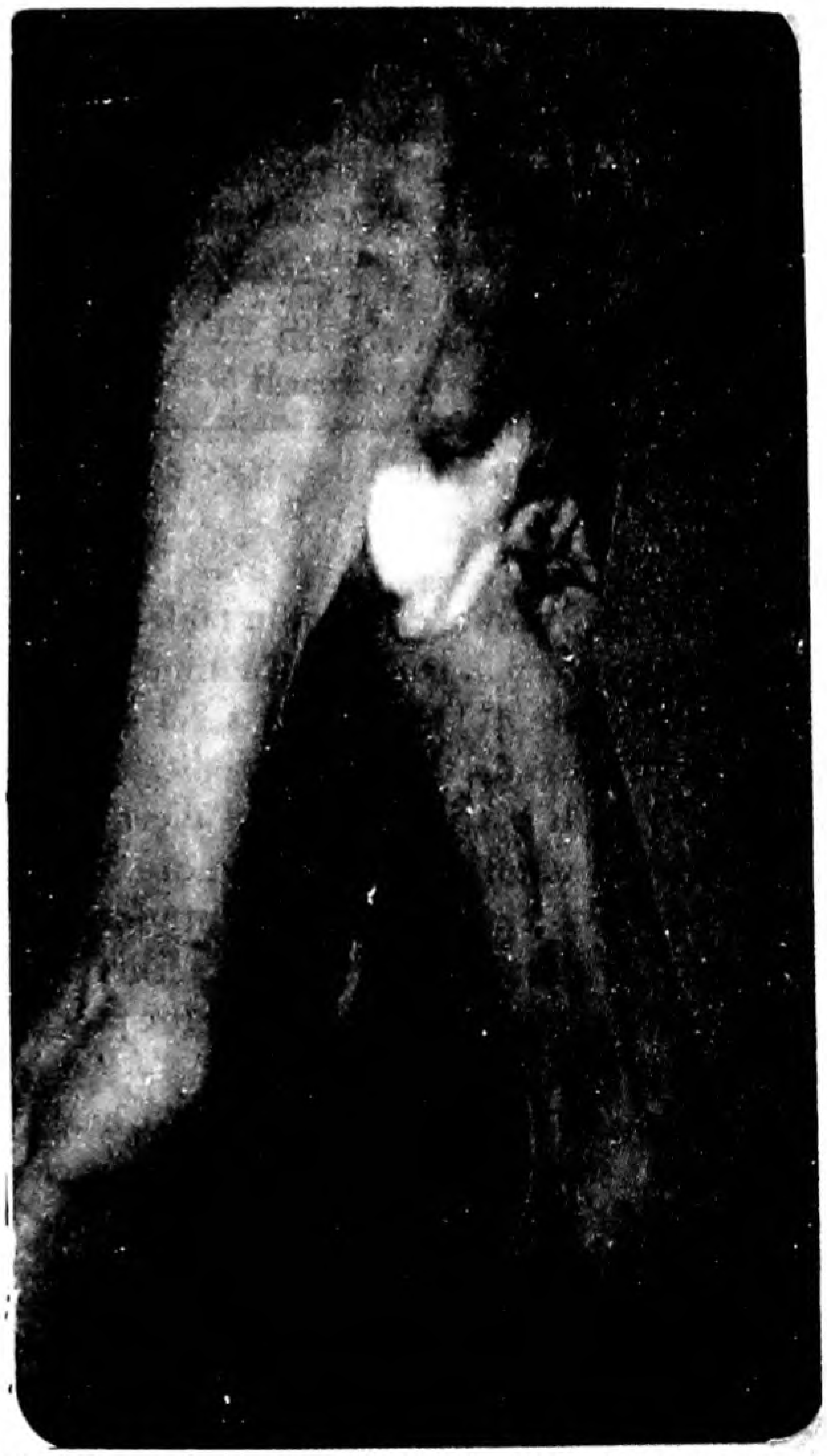

Fig.3.Fungating metastatic inguinal lympho node mass of 5 months duration.

cialities of Oncology, about I/3rd of cancers are curable, many can be prevented and the remaining can still be palliated.

It is true that cancer at some hidden sites like retroperitoneum, pancreas etc. because of their inaccessibility to examination may be detected late. But, in our set up, even the malignancies which are easily palpable or even visible are being overlooked and present in stages beyond even palliation. In this context, our observations are in disagreement to Sindney et al [3], who are of the opinion that natural history of some cancers in a important factor. The summery and conclusion of our study are given in (Inset-1)

Inset-1

- ***Mass education for early detection and diagnosis of cancer***
- ***Use of mass media to counter patient ignorance***
- ***Cancer ... though less common... to be thought of in all age groups.***
- ***Brief oncology (internship posting) to be introduced at undergraduate level***
- ***Never overtreat a patient........overtreatment is maltreatment.............***
- ***Nothing is hopeless. Proper referral at proper time ....can save a life.***

"People remember God and doctor only when they are in trouble". This maxim though basically criticizes attitude of common man, it also equates the doctor with God. Though no doctor would like to be equated with God, it is true that people do look upon, medical profession as the divine one and in their eyes the doctor's place is second only to God and doctor is looked upon by the common man as the only hope when a patient is hanging between life and death[4]. The results of our study reveal an unfortunate indictment of this divine figure in the delay for cancer treatment. Mass community level programmes for the practicing doctors at various rural and district levels seem to be mandatory for educating the practitioners in this regard.

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Original Article

Immunofluorescent Microscopic Patterns of Nephrotic Syndrome

*Parvaiz Ahmad Koul, G H Malik, *AGS Pathania, *Tanveer Riyaz.*

Abstract: Sixty five biopsy specimens from patients with nephrotic syndrome were subjected to immunofluorescent microscopy. Of 19 cases with MCGN, 16 had no deposits while 3 had mesangial IgM. Focal deposits of IgM, IgG, IgA and C$_3$ were observed in mesangial and sclerotic areas in FSGS. In MSGN, mesangial fluorescence with IgM, IgG and C$_4$ were observed. Membranous deposits in a coarse, diffuse and granular pattern was seen in cases of memb. GN, and DPGN. Diffuse linear deposits of IgG, IgA, IgM, C$_{3, 4}$ and fibrinogen were seen in patients with crescentic GN and lupus nephritis. C$_4$ fluorescence was observed more frequently than previously observed. *JK Practitioner 1995;2:169-73.*

Key Words: Immunofluorescence, Nephrotic syndrome, Immunoglobulins, Complement

Abbreviations:*GN-Glomerulonephritis, MCGN-Minimal change GN, FSGS-Focal Segmental glomerulosclerosis, DPGN-Diffuse proliferative GN, MSGN-Mesangioproliferative GN, CGN-Chronic GN, Cr.GN-Crescentic GN, Memb.GN-Membranous GN, MPGN-Membranoproliferative GN.*

Introduction

Knowledge of the changes present in glomerulonephritis (GN) has increased considerably with contributions from electron microscopy (EM), immunofluorescence (IF) and immunology. Normal looking H&F preparations may be associated with striking changes or EM & IF, and similar morphologic appearances in H&E preparations may be associated with different EM and IF patterns [1]. EM & IF and the analysis of patterns of immunoglobulins (Ig), complement (C) deposition provide important clues, though not mutually exclusive, to the underlying pathogenetic mechanisms for GN. Glomerular reactions are divided primarily on the site and pattern of complex glomerular basement membrane (GBM) or capillary wall antigens is often associated with linear deposits of Ig, so long as the relevant antigens are distributed uniformly along the glomerular capillary walls [2]. However, if the circulating antibodies are directed to other nature or planted antigens that discontinuously distributed along the capillary wall or in the mesangium, other patterns of Ig deposition may result. Disease associated with the deposition of circulating antigen-antibody complexes (immune complexes) in the glomeruli characteristically are associated with granular deposits of Ig, varying in location from subepithelial to subendothelial to pure mesangial deposits [3,4]. These pattern may be distinctive or merge with one another.

IF microscopy is performed on all renal biopsies to complement routine histopathology. The present study presents the IF spectrum of 65 cases of nephrotic syndrome and reviews the available literature over the IF microscopic patterns of deposition in various causes of NS.

Material and Methods

Sixty five adequate kidney biopsies from patients with nephrotic syndrome were included in the study. The clinical and light microscopic details of these cases have been described elsewhere [5]. Included were 19 cases with MCGN, 9 cases with FSGS, 8 cases with MSGN, 4 cases with Memb. GN, 12 cases with DPGN, 6 cases of MPGN, 3 cases of lupus nephritis, 2 cases of crescentic GN and 1 case each of focal prolif. and chronic GN. Serum C$_3$ complement levels were analyzed in all cases. Fresh biopsy specimens were snap frozen and sections cut in cryostat. After embedding in OCT fluid, staining for direct fluorescence was done using fluorescin tagged antihuman IgG, IgM, IgA, C$_3$, C$_4$ and fibrinogen.

Results

Of the 19 cases with MCGN, 16 exhibited no deposits whereas faint, irregular, granular mesangial

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deposits of IgM (n=3), IgG (n=2), C_3 (n=1) and fibrinogen were demonstrable in the others. One patient also demonstrated arterial deposition of IgM & C_3. Serum complement levels were observed to be normal in all except one case, mean levels being 84.78±23.6mg/dl(range 22-130mg/dl).

Of the 8 patients with FSGS, one patient had no deposits, whereas focal granular deposits of IgM (n=7), IgA (n=3), IgG (n=4), C_3 (n=5), C_4 (n=1), and fibrinogen were observed in mesangium and sclerotic areas of the glomeruli in others. One patient each had arterial and tubular basement membrane fluorescence with IgG and C_3. Complement C_3 levels were normal in all except one case (mean 96.2±33.9, range 46-169mg/dl).In cases of DPGN, dominant fluorescence with IgG and C_3 was observed in a diffuse generalized granular form. 8 cases had membranous deposits with IgG and C_3. Mesangial deposits of IgM, IgA and C_3 were also observed (n=1). 4 cases had epimembranous deposition of IgG, IgM and C_3. A linear pattern of deposition in an interrupted manner was observed in one case in the membrane. Arterial and tubular basement membrane deposits were found in 2 & 1 case respectively. Low levels of C_3 were seen in 4 cases, being normal in the rest(mean 64±42.03mg/dl, range 23-180).

Diffuse generalized granular deposits of IgA, IgM (n=4), C_3 (n=7), C_4 (n=2) were observed in mesangium in MSGN (n=7). 2 cases had associated membranous deposition with IgM, IgG and C_4. C_3 levels (81.4±30.7, range 11.5-106mg/dl) were low in two cases but were normal in the rest. Membranous deposits were observed in a coarse, diffuse and granular pattern in all cases of Memb. GN. While the deposition consisted of IgG and C_3 uniformly, IgM (n=2), IgA (n=1) and C_4 (n=1) deposition was also noticed. C_3 levels were normal in the cases.

Membranous deposits of IgG and C_3 were found universally in cases with MPGN in a diffuse generalized and granular pattern. Mesangial deposits of IgG and IgM with arterial C_3 and IgA were seen in two cases. C_4deposition in mesangium was observed in one patient. C_3 levels in these cases demonstrated uniform hypocomplementemia (mean 23.25±10.09, range 23-41 mg/dl).

Diffuse, linear deposits of IgG, IgA, IgM, C_3, C_4 & fibrinogen were seen in both the patients with crescentic glomerulonephritis. One patient had associated membranous fluorescence with IgG and C_3 and another had similar fluorescence in mesangium. Both cases had demonstrable hypocomplementemia (47,53 mg/dl).

In the 3 cases of lupus nephritis, 2 cases had diffuse heavy, granular deposition of IgG, IgM in the membrane and mesangium. Epimembranous deposits were observed in 2 cases and consisted of IgG, IgM and C_3. In the other case of lupus nephritis, the deposition of IgG, IgM, C_3, C_4, fibrinogen was noticed in the membrane, arteries and tubular basement membrane in a linear pattern. C_3 levels ranged from 7-24 mg/dl (12.3±8.6) with low levels in all the 3 cases.

Mesangial deposits of IgA, IgG and C_3 were seen in a generalized granular fashion in the patient with chronic glomerulonephritis, whereas membranous and arterial linear deposits of IgG and C_3 were noticed in the patient with focal proliferative glomerulonephritis. C_3 levels were normal in both the cases of FPGN and ch.GN.

Discussion

Majority of our patients with MCGN had no deposits. Immunofluorescence findings in this disorder are noteworthy for the lack of deposition of Ig and complement in glomeruli [4,6-10]. Although some investigators have described the occasional presence of small flecks or coma-shaped deposits of IgM in the mesangium, especially at the vascular pole, and IgG and C3 in an amorphous pattern in the mesangium, the definition of an uncomplicated minimal change lesion, in light of present knowledge, should include the complete absence of immunoglobulin and complement deposits [11]. However, the presence and significance of Ig deposits in minimal change lesion remains controversial. 3 of our patients also had faint deposits of IgM in a granular fashion. Prasad and co-workers and others have identified three groups of patients with "minimal change nephrotic syndrome": one with negative immunofluorescence,a second with immunoglobulin deposits (usually IgM) in a mesangial distribution and a third with C3 deposits with or without accompanying immunoglobulin (again IgM in 8 of 10 cases) [12]. Despite the heterogeneity of immunofluorescence microscopic appearance, the authors indicated that all three groups of patients had similar clinical courses and responses to therapy and believed that immunoglobulins did not have a pathogenetic role. The alternate view, held by other investigators [13], is that glomeruli with IgM deposits and morphology characterized by varying degrees of mesangial changes (including changes so mild as to permit categorization of

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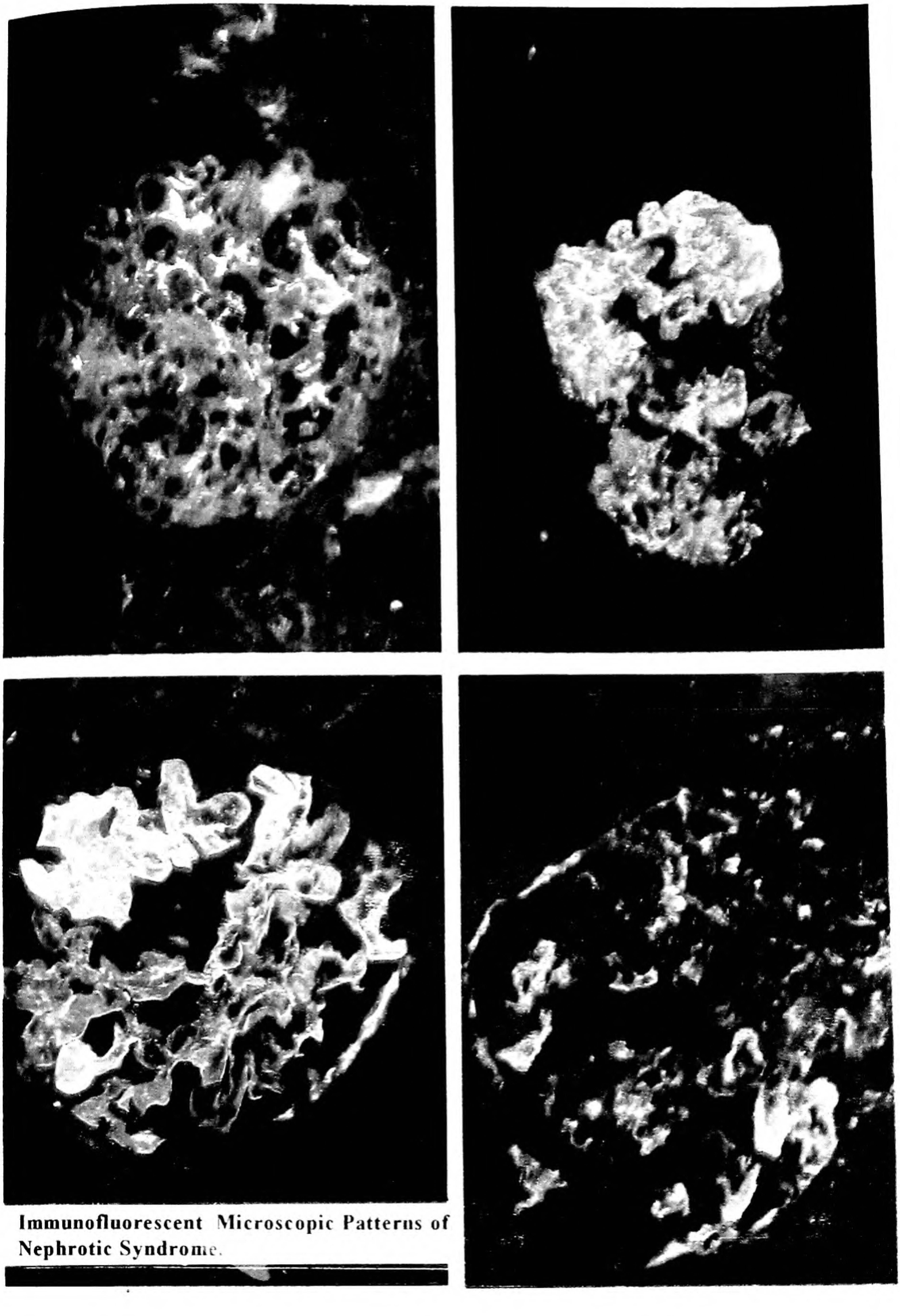

Immunofluorescent Microscopic Patterns of Nephrotic Syndrome.

the biopsy into a minimal change lesion group) in patients with nephrotic syndrome are part of a distinctive clinical-pathological entity that has tentatively been termed IgM mesangial nephropathy. None of our cases could be categorized as IgM nephropathy.

4 of our patients with MSGN had a concomitant deposition of IgA and IgM with C_3 in the mesangium. The deposition of IgM was not overwhelming in any case to justify classification as IgM mesangial nephropathy. A frequent, although not invariable, finding in MSGN is the presence of IgM deposits primarily in the mesangium, but also occasionally as small granularities, in the capillary walls [6,14,15]. The pathogenetic role of such deposits is unknown. In many instances, C3 has been observed in a similar distribution, although not in all series, IgG and IgA are variably present and usually with a lesser intensity than IgM and C_3.[1,6,]. By definition, predominant IgA deposits result in categorization as Berger's disease. One of our patient had a predominant deposition of IgA, C_3 & C_4 which would possibly merit classification as IgA nephropathy. The patient had clinical hematuria with normal renal function. He had a poor response to steroids.In several series the immunofluorescent findings have been reported as negative for Ig deposits in most cases [1,17]. They presumably represent instances of minimal change disease with more prominent mesangial cell hypercellularity.

Of our patients with FSGS, Ig & C_3 deposits were observed in sclerotic and mesangial areas. IgM, Clq, and C3 deposition has been reported in an irregular, granular, or nodular distribution only in association with the focal sclerotic lesions [1,18]. Unaffected glomeruli on occasion may contain variable amounts of IgM and C3 in a mesangial distribution [18]. Diffuse, linear IgG deposition may be noted in absence of circulating anti-GBM antibodies [1],the pathogenetic importance of the linear deposits of IgG being unknown, but probably related to nonimmunological factors. One of our patients also had arterial and basement membrane fluorescence with IgG & c_3[19]. The mesangial deposition of IgM and C3 in a granular pattern suggests an immune complex disease. It is also possible that a primary disorder of the capillary tuft leads to nonspecific trapping of IgM with subsequent binding of C1q and C3 [1].

Granular, membranous deposits of IgG and C_3 were observed in all our patients with Memb. GN with associated fluorescence with IgM, IgA & C_4 in some cases

Immunoglobulin G is nearly always present in a uniform granular distribution outlining all of the capillary loops but sparing the mesangium [20-2]. In early cases, the deposits are small and may have a "linear" or continuous appearance at lower power magnification. In advanced cases, the deposits are larger and still uniform in size, but focally may show coalescence. In still advanced cases, or after prolonged remission of proteinuria, the IgG deposits may be weak and irregular or even negative, corresponding to the decreasing electron density seen by electron microscopy. IgM and IgA deposits have been reported to be scanty. C3 usually is present in a pattern and intensity similar to IgG. [21,22]. One of our cases had C_4 deposition. In MPGN type I, all investigators describe prominent C_3 deposition in an irregular and granular distribution, tending to outline the periphery of the lobule, with variable localization in the mesangium [1,6,23]. IgM and IgG, have been in an inconsistent and segmental granular capillary wall and, less often, mesangial distribution but are not demonstrated in as many as one-third to one-half of the cases[23]. Our patients had a uniform distribution of IgG and C_3. IgA may be present in approximately 33 percent of the specimens. The early acting complement components such as Clq and C4 are found only slightly less often than C3, usually in association with prominent Ig deposits. Although no clear-cut difference in clinical course has been demonstrated, some authors have divided Type I MPGN into sub-categories, depending on the type of immunofluorescence patterns: Type A, C3 only; Type B, C3 predominant with some IgG or IgM; Type C, C3 and Ig in equal intensity. Each may have differing degrees of mesangial or peripheral capillary wall involvement[23]. In type II MPGN , most commonly, C3 observed in a discontinuous linear ("railroad track") pattern outlining the capillary walls, Bowman's capsules, tubules, and occasionally arterioles in a distribution identical to the basement membrane abnormalities. In addition, there may be granular C3 deposits in may capillary walls

Figures (1-4)(on facing page). Fluorescence photomicrographs showing (1)Mesangial fluorescence with IgG, (2)Membranous and mesangial fluorescence with IgM, (3)Membranous and mesangial fluorescence withIgG and, (4)Membranous fluorescence with C3.

and large globular masses of C3 in many mesangial areas [24]. Hypocomplementemia was observed in all of our cases. It has been reported earlier in upto 70% cases of MPGN.[1,24]. Kim and co-workers[24] demonstrated that the largely linear capillary wall staining was due to the presence of two very closely approximated parallel lines ("railroad tracks") of C3, which, when compared with the same preparations stained with periodic acid-Schiff, was present on both sides of the basement membrane, rather than within it. They also described the presence of "mesangial rings" corresponding to the granular deposits in the mesangium. The fluorescence patterns of other glomerulo-nephritides were similar to those reported earlier [25-28].

Pathogenetic information about different forms of glomerular disease, identifying them as immune complex or anti-GBM mediated diseases is obtained by using IF microscopy and hence IF microscopy should be employed routinely in evaluation of all biopsies. However, the response to therapy vis a vis various IF microscopic patterns needs to be studied subsequently.

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Early Structural Changes in Rat Liver After Lithium

MS Shah, Ghulam Hassan

Abstract: Effects of ingestion of lithium carbonate for 90 days on the liver of 20 albino rats was studied, comparing with 10 controls. Gross changes observed ranged from a mild decrease in weight to cyst formation. Microscopy of the liver revealed loss of normal cellular architecture, degenerative and cystic changes and Kupffer cell hyperplasia. *JK Practitioner 1995; 2:174-5.*

Key Words : Lithium, Liver, Hepatotoxicity

Introduction

Since Cade first reported the use of lithium as an antimanic drug in 1949 [1], it has become one of the most valuable drugs in the treatment of affective disorders. In 1970 FDA approved the use of lithium for treating acute mania and as a prophylaxis against recurrent mania. A light microscopic study of lithium treated kidneys in dogs by Radomski et al in 1950 [2] showed degenerative changes in its various parts which led to the possibility of affliction of other tissues by the drug.

Material and Methods

Thirty albino rats weighing 150 gms on an average were used during the present study. The animals were categorized in A and B groups. Ten animals in groups A received usual food and plain water and served as controls, whereas 20 animals in group B in addition to usual food and drinking water received lithium carbonate in a dose of 4 gm per 100 gms of body weight mixed with flour in the form of pellets daily for 90 days. After 90 days, the animals were killed and the liver specimens obtained were processed for histopathological examination. Staining was performed by Haematoxylene and Eosin stain.

Observations

Gross examination of normally lobulated liver from drug treated animals showed various changes which included a mild decrease in weight and color changes varied from reddish brown to grayish one. Appearance of whitish spots and cyst formation on the surface of lobulated liver were also apparent [Fig.1].

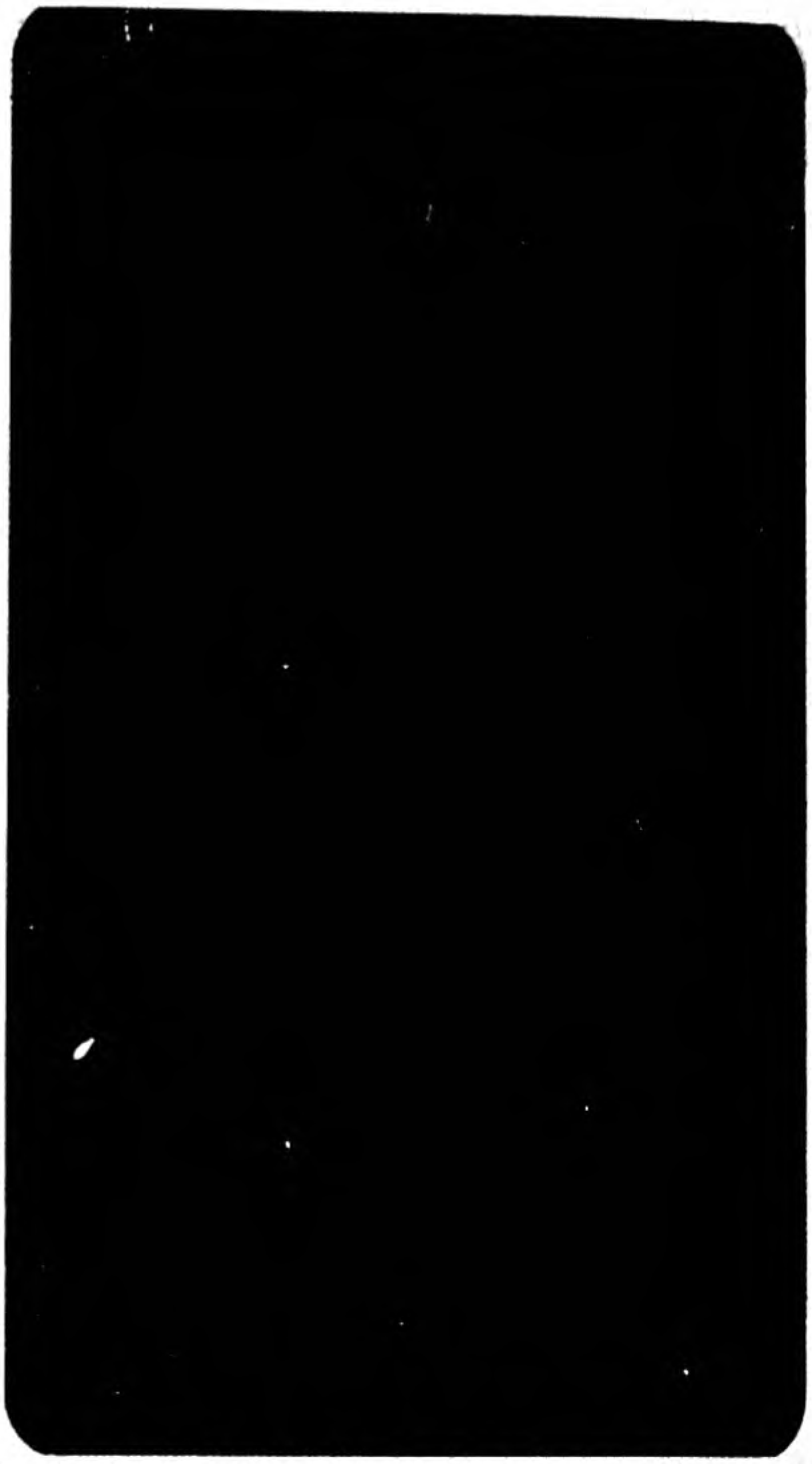

Fig. 1. Photograph showing cyst formation in liver on gross examination

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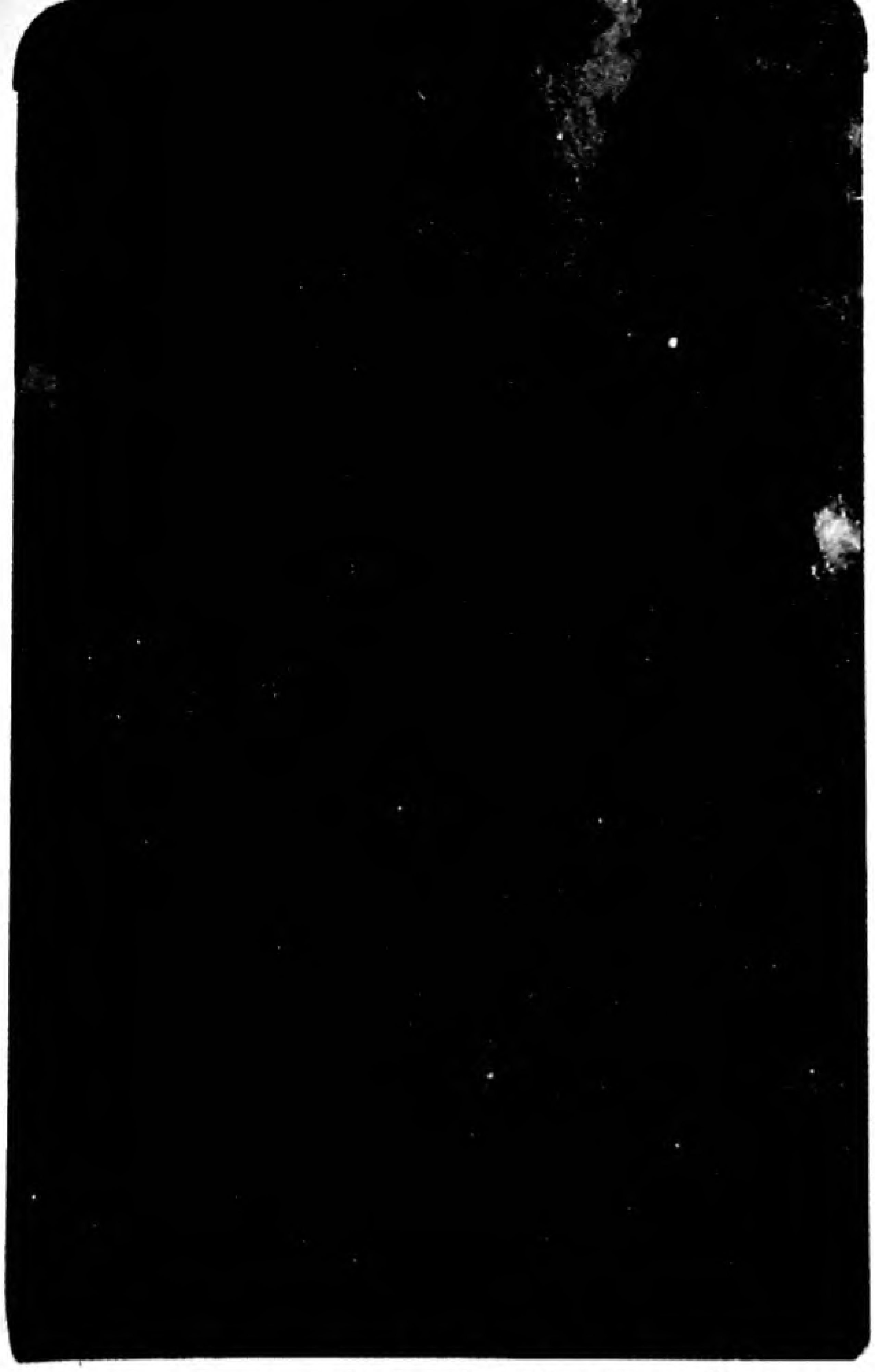

Fig.2. Microphotograph showing Kupffer cell hyperplasia (arrow) & cytoplasmic degeneration(CD) H&EX40.

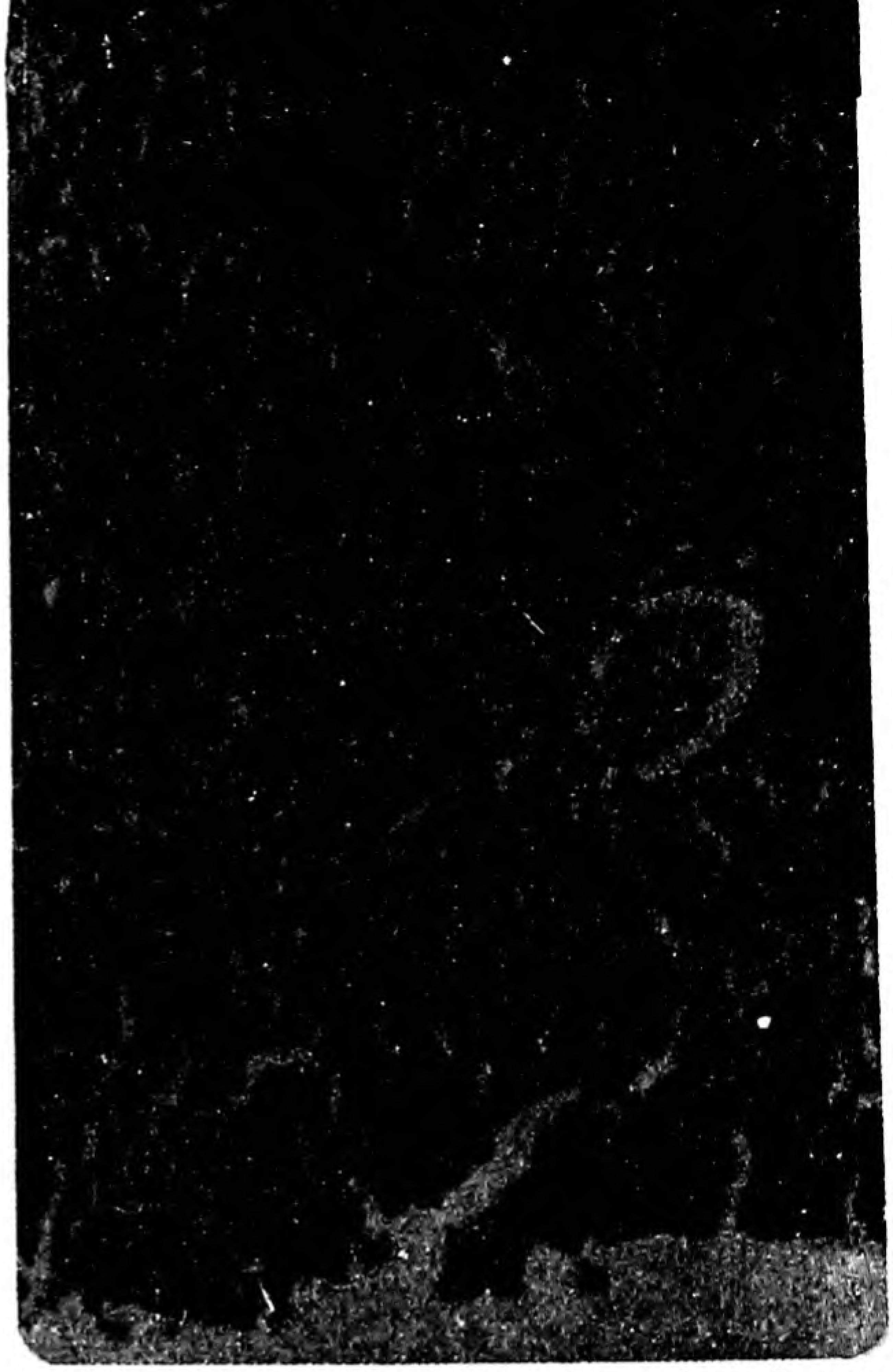

Fig.3. Microphotograph showing congestion of central veins (C), liver cell degeneration (D) and increased sinusoidal spaces (S). H&EX10.

Microscopically hepatocytes showed an indistinct cellular outline with cytoplasmic degeneration associated with loss of normal cellular architecture in drug treated animals. Focal areas of necrosis followed by cyst formation was also observed [Fig.2]. In addition to distortion of cellular configuration, a mild increase in spaces between sinusoids was also obvious. There was concomitant increase in Kupffer cells and congestion of central veins [Fig.3]. Areas of vacuolations were also seen occasionally.

Discussion

The study of the effects of lithium on liver so far has not received much attention. Nordenberg et al 1981 [3] determined the effects of lithium on various enzymatic levels on liver. They observed a marked inhibition of such levels.

In the present study, liver after treating with lithium showed indistinct cellular outline with cytoplasmic degeneration and loss of normal cellular architecture. In addition focal areas of necrosis, cyst formation associated with mild increase in sinusoidal spaces and kupffer cell hyperplasia were also seen. Congestion of central veins was also noticed. Thus it is concluded that liver manifests both gross as well as microscopic changes after lithium therapy. The clinical implications of these findings need to be studied in humans as well.

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In Memoriam

Late Dr.Ali Muhammad Jan, MRCP, DCh
(1914-1988)

Dr. A. M. Jan was born on 3rd September in the year 1914 in a well known family of "Fazili" dynasty. He graduated in medicine at the age of 23 years in the year 1937 from King Edward Medical College Lahore. He was adjudged the best out going and distinguished graduate of the year, besides securing the merit certificates, Dr. Khan and Dr. Nelson memorial gold medals.

Dr. Jan started his career as a Mobile Medical Officer when first posted at Kishtwar and also as Tuberculosis Control Officer at Tangmarg and his tenure of service almost overlapped whole valley till 1950 before he proceeded to England for obtaining membership and completing the D.C.H. courses in a record period of 9 months in the very first attempt, a remarkable feat by all standards. He returned from England to S.M.H.S Hospital as physician specialist and started first sick children unit under his guidance. He was appointed as Professor in 1964. During this period he created a name as a physician and a "Messiah" and even his taught made a name in the profession both in India and abroad. He believed in discipline, dedication and devotion to the profession and did not compromise on terms that he felt were not according to standards. He had a quality of not being carried away by the dictates of his superiors; this fact was mainly responsible for his premature retirement from active service.

He devoted his life as a private medical practitioner to the cause of patient care where he created a record of excellence so much so that he became the best psychiatrist for his patients and even miracles were attributed to his name. For all times to come his name will always be held in esteem. He looked unsocial, isolated and shy in public conversations but had a vast social circle and was a powerful conversationist in private. He would always help his friends, colleagues and needy patients. His hobbies included golf, fishing and chess besides devoting his time in medical and religious literature. Dr. Jan will be remembered for his meritorious services in the field of medical sciences in Kashmir and his standing contribution remains the Institute of Medical Sciences in Soura, Srinagar.

(Write up contributed by Prof. G.H.Hajini, Head, Department of Skin and V.D., Government Medical College, Srinagar, Kashmir).

What I know about the Hippocrates of the Valley?

A dedicated, disciplined, punctual to his duty at the hospital or clinic (even on holidays), never sold his prescription free or complimentary, never bothered for politicians or bureaucrats at any level and rendered equal treatment to the poor and the rich, remembered his old patients' data like a computer. His prescription had short clinical notes, investigation and diagnosis. He would be up-to-date with latest journal/books, but never paid much attention to compile his vast clinical knowledge in the form of articles in journals or write a book. However, he produced excellent students/residents who have fulfilled this area at international level. Dr. Ali Jan will remain a domestic name in the valley for many future generations.
May God give peace to his soul forever. I wish every doctor follows his honest ways of clinical medicine and dedication towards his profession and patient care.

G.M. Malik
Editor In Chief

Some Memorable Pictures of Late Prof. A.M. Jan

WELCOMES YOU

1. Dr. Jan with his colleagues during Annual Cardiology Conference (1987) at Srinagar. (L to R) Dr. P.S.Sikand, FRCS; Dr.A.M.Jan,MRCP; Dr.Sharma,MCh. and Dr. G.M.Malik,MD

Dr.Jan during the International Cardiopulmonary Conference at Srinagar (1985), (L to R) Dr.G.Q.Allaqaband (Course Director), Dr.A.M.Jan (Vice Chairman SKIMS), Dr.B.K.Anand (Director, SKIMS), Dr.Girja Dhar(Principal Govt.Medical College,Sgr) Mr.Jagmohan(Governor, J&K State), Dr.S.Z.Ahmad (Course Director) and Dr.G.M.Malik (Coordinator)

(Sponsored by American College of Chest Physician) [Courtsey - Prof. F.A. Khan, Course Director, USA)

Clinical Images

Peutz - Jegher's Syndrome

(JK Practitioner 1995;2:178)

SA. Kadla, Javed Basu, GM Malik

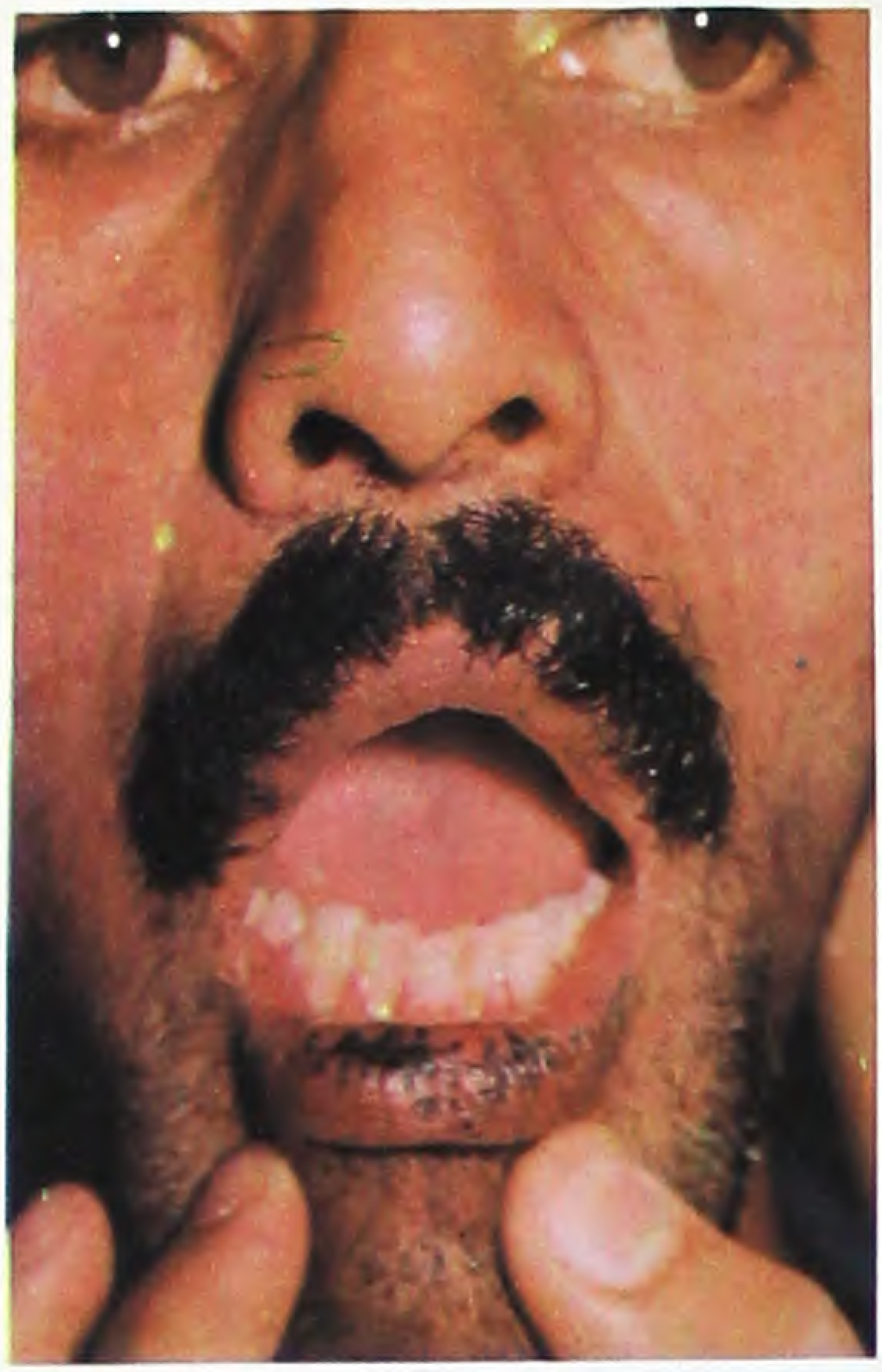

Fig.1

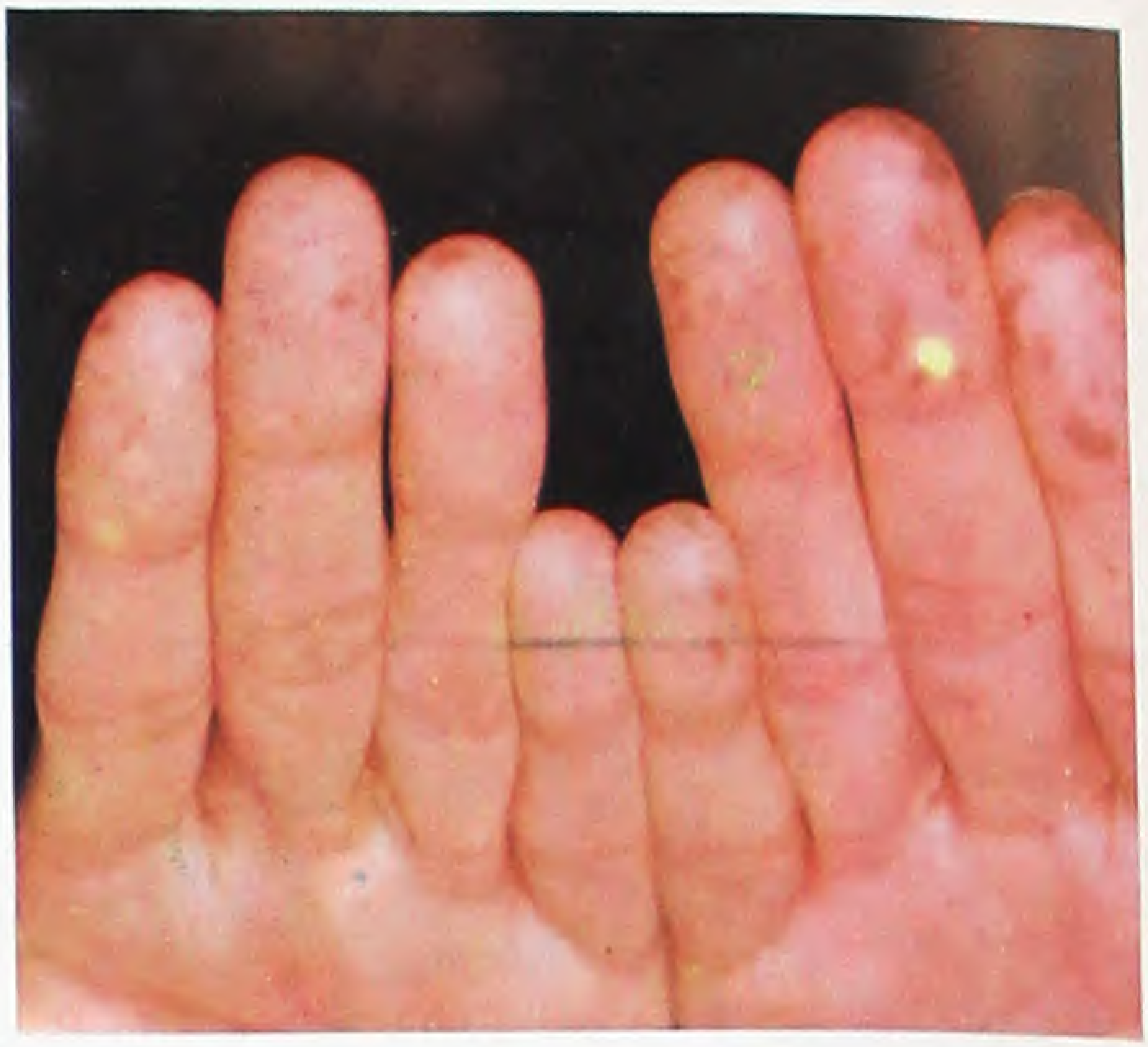

Fig.2

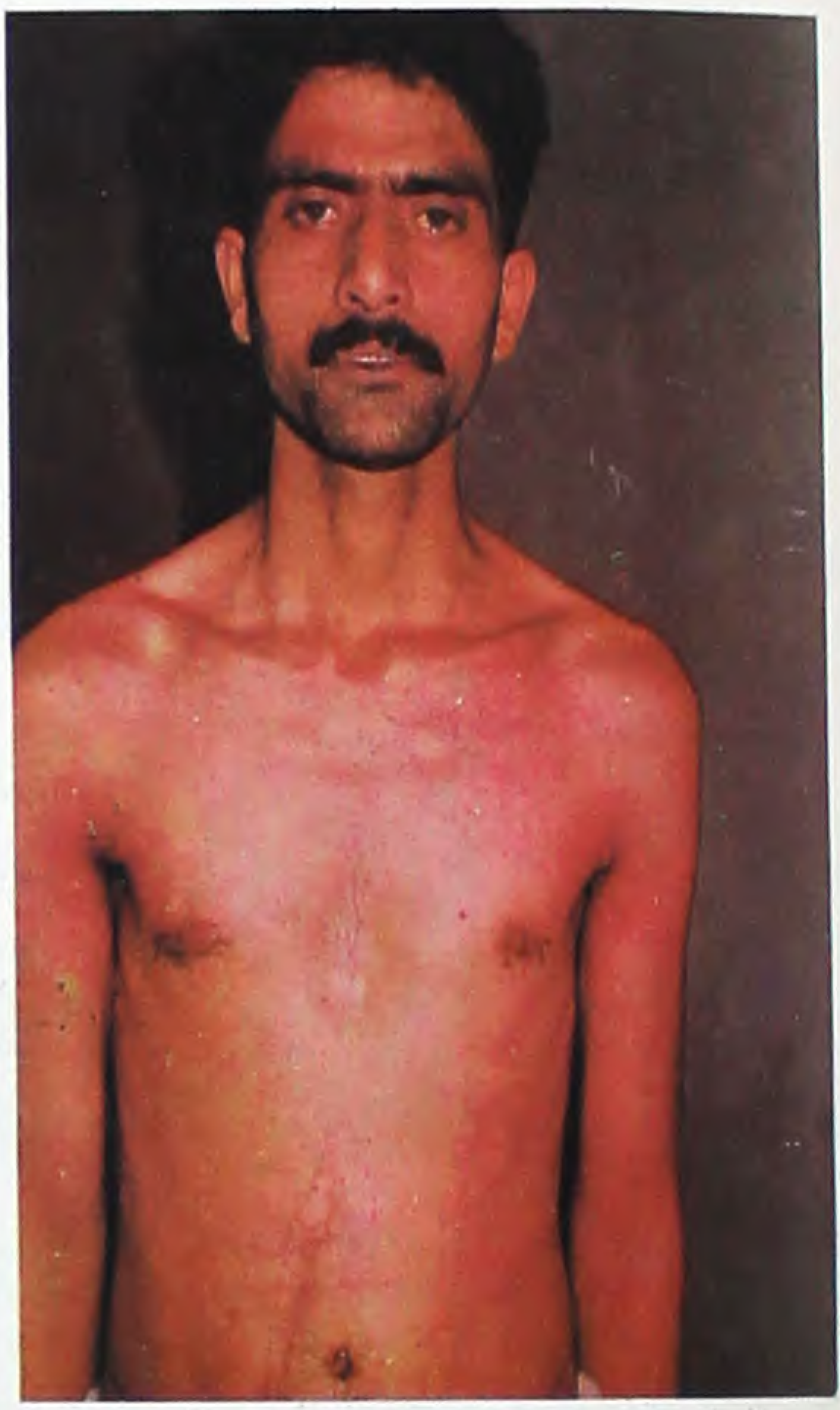

Fig. 3

Figs. 1&2. A young man with pigmentation of lips and hands.
Fig. 3. Brother of the patient with lip pigmentation and a surgical scar over abdomen (operated for small intestinal polyps).
Dignosis: A family with Peutz - Jegher's syndrome.
(Rare familial syndrome of polyposis of small intestines with pigmentation of skin and mucus membranous, transmitted as autosomal dominant trait)

Division of Gastroenterology, Department of Medicine, Government Medical College, Srinagar.
Correspondence to: Dr. G.M.Malik, Head, Division of Gastroenterology, Deptt. of Medicine, Govt. Medical College, Srinagar, Kashmir, Pin-190 009. *Accepted: August 31, 1995.*

Roentgenogram of the Month

A young male with muscle weakness and a mediastinal mass.

(JK Practitioner 1995;2:178-81)

Parvaiz A Koul, Mansoor A Khan, A Wahid.

A 32 year old male presented with a 10 day history of progressive weakness of upper limbs and an 8 day history of drooping of right eyelid. There was no history of a preceding febrile illness, drug intake, any previous such episodes, bowel/bladder or sensory disturbances. Clinical examination revealed an average built young man having grade III-IV weakness of proximal muscles of all the 4 limbs. The distal musculature, trunk and neck muscles and sensory system were normal. Rest of the general physical and systemic examination was normal. A 3-minute upward stress of the eyelids revealed a bilateral droop. The hemoglobin was 13.9 g/dl, the TLC 6.6×10^9/l with 66% polymorphs, 26% lymphocytes 8% monocytes, the platelets 214×10^9/l with a normal peripheral blood smear. The serum urea was 33 mg/dl, the creatinine 0.92mg/dl, the glucose 104mg/dl, the calcium 9.02mg/dl, the phosphorus 3.5mg/dl, the sodium 129mmol/l, the potassium 4.2mmol/l, the bilirubin 0.8 mg/dl and the liver enzymes were normal. CK was 98IU/l and LDH 550 IU/l. ECG was normal. Latex fixation test was negative and no autoantibodies could be detected. Immunoglobulin concentration was normal. The chest x-ray is shown in Fig 1 and a CT scan of the chest in Fig 2. Electromyographic studies revealed a decremental response of the action potential after a few stimuli further followed by an incremental response; which was reversed by edrophonium infection. Nerve conduction studies revealed normal conduction velocities in all the 4 limbs.

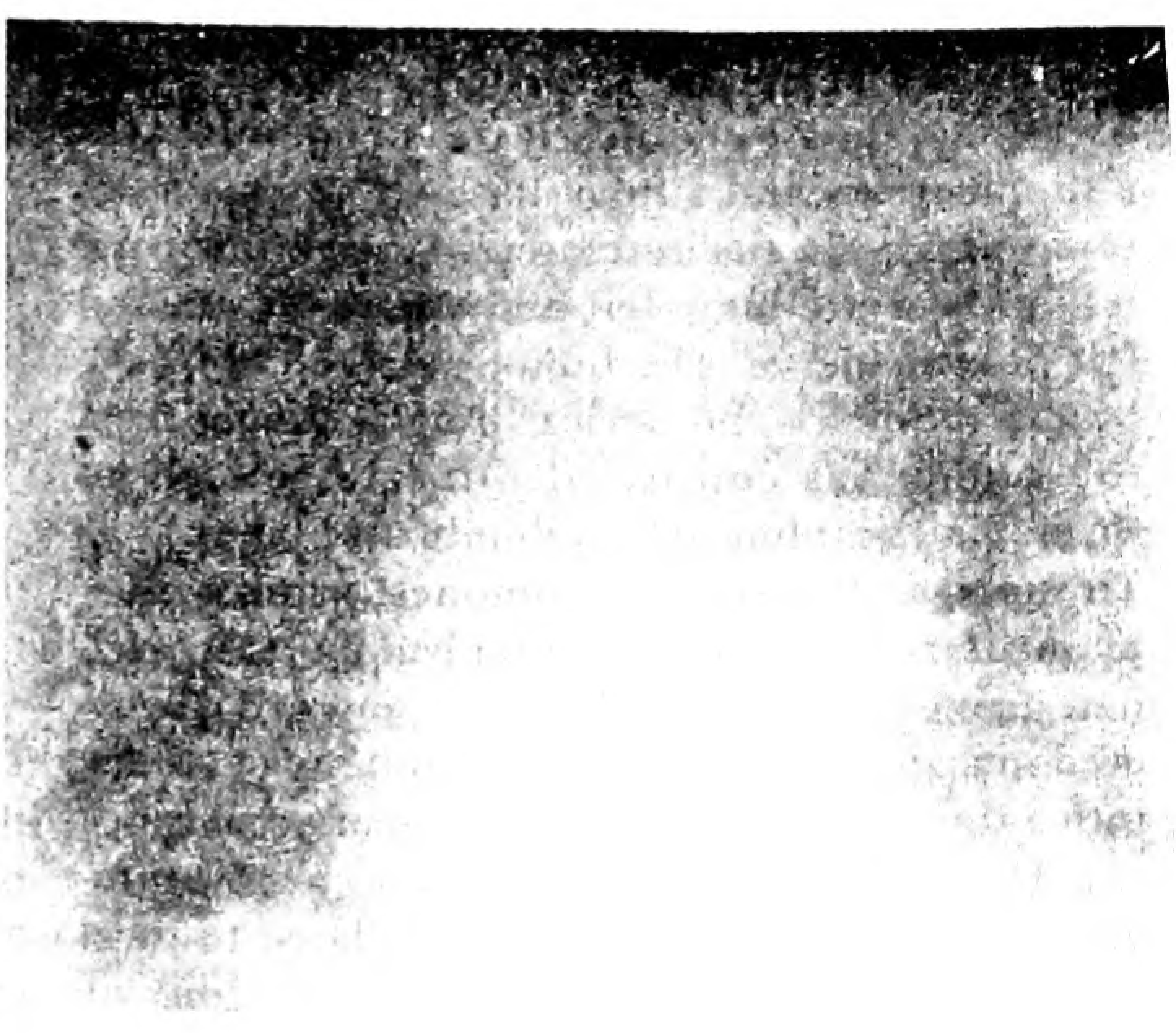

Fig.1

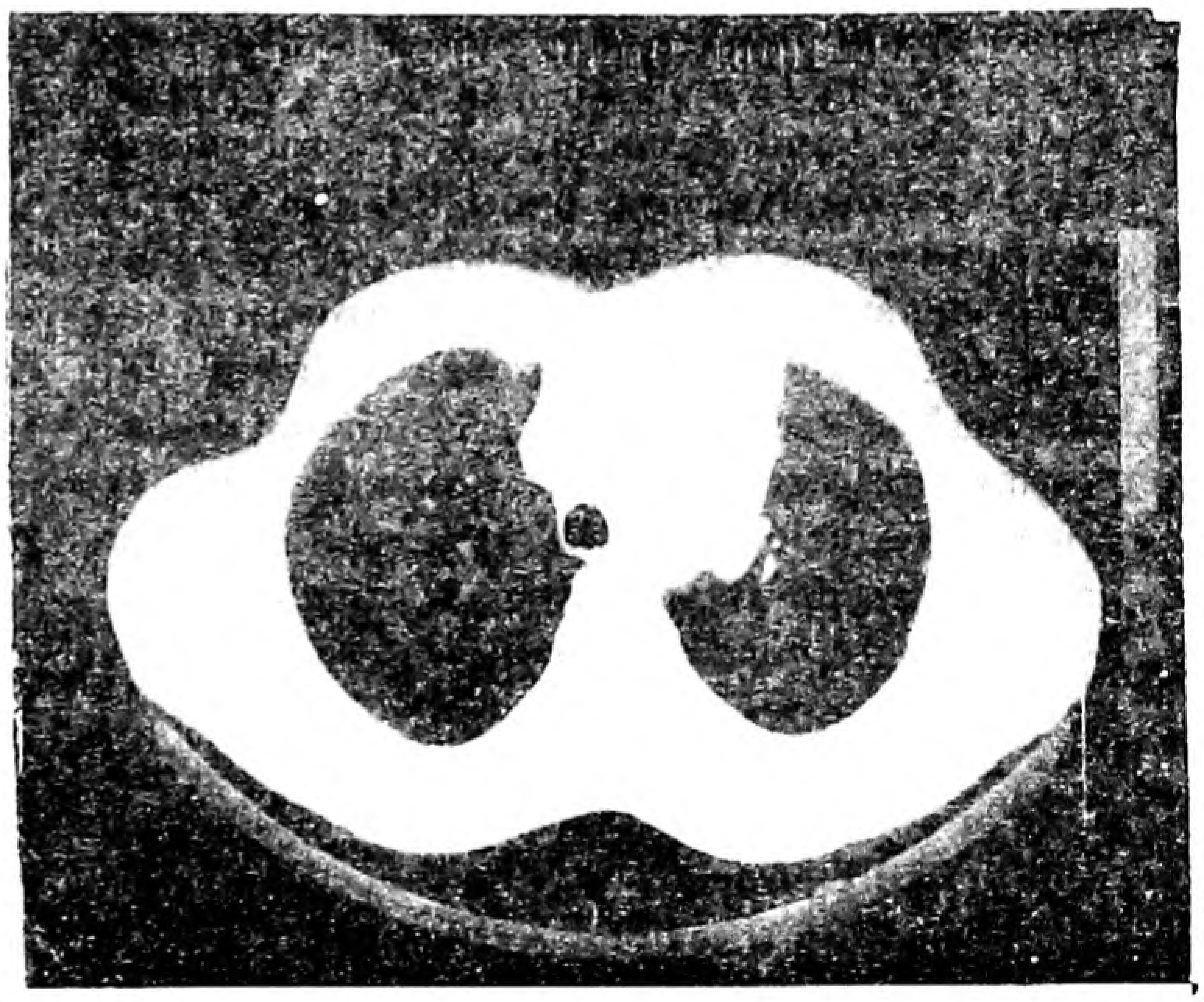

Fig.2

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Diagnosis: Malignant thymoma with myasthenia gravis.

Radiograph of the chest revealed a mediastinal mass. CT chest revealed a large thymic mass in the anterior mediastinum. FNAC from the mass showed sheets of mature lymphocytes with sprinkling of immature lymphocytes and histiocytes. CT guided percutaneous needle biopsy showed sheets and masses of normal looking lymphocytes with occasional Hassal's corpuscles. Patient underwent a thoracotomy and a huge hard thymic mass was seen occupying the retrosternal space involving left apex of the lung, left pulmonary atrium and left phrenic nerve. The tumor was seen to be adherent to pleura and pericardium. A biopsy from the tumor was consistent with a diagnosis of thymoma (lymphocytic predominance type).

Thymomas, the second commonest primary neoplasm of the mediastinum (after lymphoma), originate from thymic epithelium [1] and are discovered in majority of the cases in middle aged adults, with a slight female preponderance of occurrence [2]. The patients at presentation may be asymptomatic or may manifest symptoms related to local structures (due to compression or invasion) or may have systemic autoimmune disease. Hematologic abnormalities, especially pure red cell aplasia are also present in some cases [3].

Myasthenia gravis is the most common autoimmune disease associated with thymoma. About 15% patients with myasthenia have this tumor [4-6], and about 35% (7-54%) of patients with thymoma have myasthenia [5,7]. Myasthenia may be observed concomitant with red cell aplasia [3]. Other autoimmune conditions associated with thymoma include hypogammaglobulinemia [8], systemic lupus erythematosus, rheumatoid disease, polymyositis, Grave's disease, inflammatory bowel disease, pernicious anemia, Sjogren's syndrome, pemphigus vulgaris, and alopecia areata [5,9,10].

Roentgenographically thymomas are round or oval and their margins are smooth or lobulated [11]. Protrusion to either side may result in the displacement of adjacent structures. When very large in size, their site of origin may be exceedingly difficult to establish short of arteriography. CT is the imaging procedure of choice and should be employed routinely. CT demonstrates the lesion clearly, demonstrates anatomic relationships, and adherence and invasion of the adjacent structures. In a study of 16 cases, CT demonstrated the lesion clearly in all except two cases in whom invasive thymoma showed a markedly irregular and infiltrative pattern on CT [12].

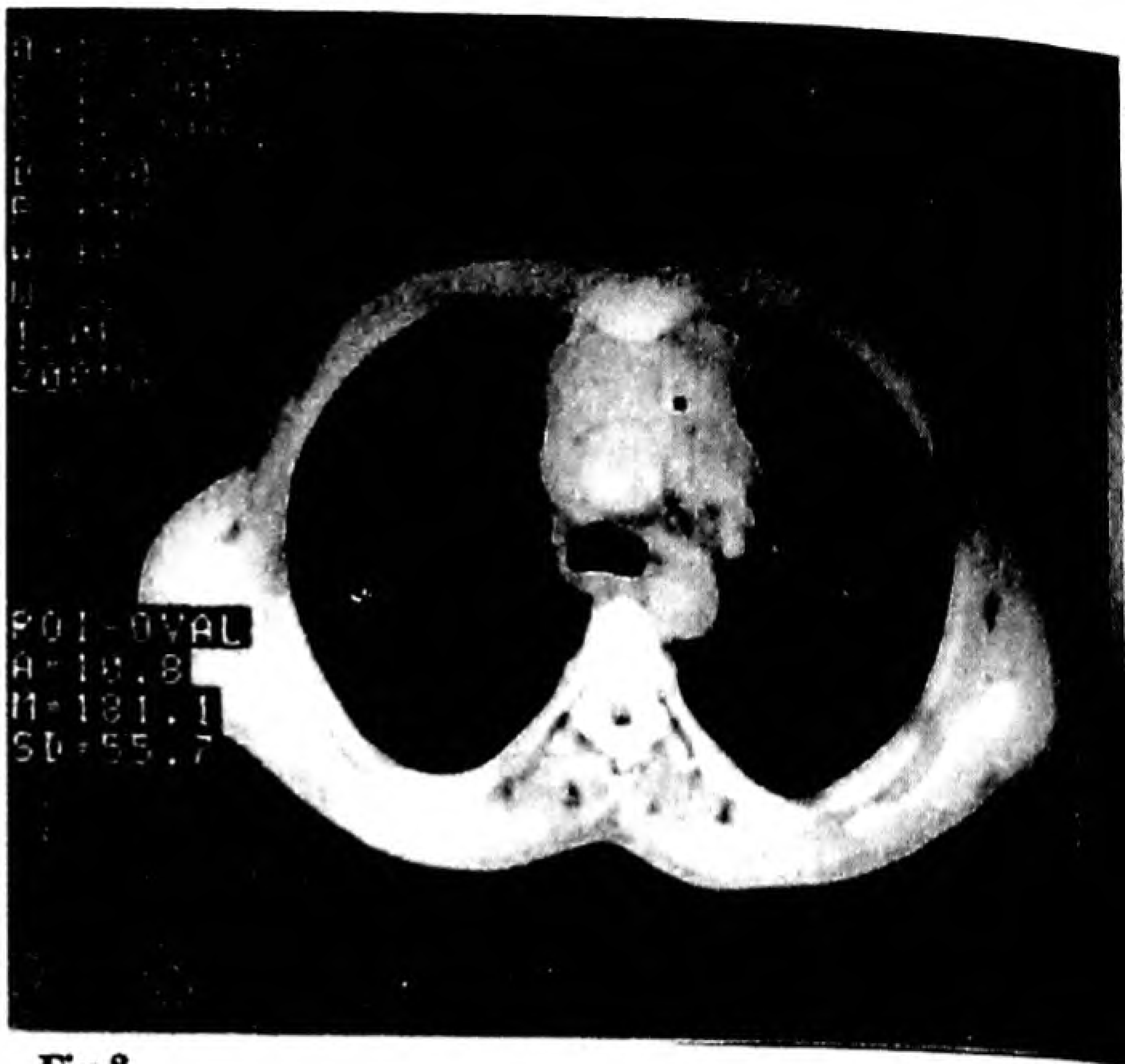

Fig.3

Pathologically, the tumors present as well encapsulated, round or slightly lobulated masses, ranging from 5 to 15cm in diameter and foci of calcification, hemorrhage and necrosis may be seen [5]. Infiltration of adjacent structures occurs in 10 to 15% cases [5]. Histologic patterns are variable but the most commonly employed classification consists of predominantly lymphocytic, mixed lymphoepithelial and predominantly epithelial forms. The neoplastic cells in the ones with epithelial element may be polygonal or spindle shaped [2,5,9,10]. A characteristic feature of all the three varieties is the presence of perivascular spaces, which are centered about small blood vessels and contain proteinaceous fluid and scattered lymphocytes.

Majority of thymomas are slowly growing and cure results from surgical excision; however, they can be locally invasive or may recur after excision. Local invasion varies from 7-50% [13] and recurrence in them is a rule as total surgical excision is not possible [5]. Extrathoracic metastasis are uncommon [5].

Our patient had a locally invasive inoperable tumor. After surgery he received radiotherapy and is now 4 years on follow-up, needing only neostigmine. There is no evidence of a further spread of the tumor.

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New Horizons in Ophthalmology

Lasers in Ophthalmology

(JK Practitioner1995;2:182-3)

*Bashir Ahmad, **AR Nasti, ***Mehmooda Ashai, ****Tariq Qureshi.

Laser is a coherent, calumniated and concentratedbeam of mono-chromatic light with an ability to focus to a very small spot size. The development of ophthalmic lasers has had a tremendous impact on our ability to preserve or restore vision by providing an effective treatment which is easy to administer and has less complications.

The concept of retinal photocoagulation was first introduced 40 years back by Meyer Schwickorath (1949) who focussed rays of sunlight for retinal photocoagulation. In 1963 ruby laser was used clinically for the first time for retinal photocoagulation followed by introduction ofYAG laser and carbon dioxide laser in 1971 while in 1972 Krypton laser was introduced. The continuous wave tunable organic dye laser was introduced in 1951.

In recent years the progress in technology has led to development of new generation diode laser. Diode laser has the advantage, that the size of light emitting region is in millimeter range so a very compact air cooled laser system has been developed.

Lasers are also used for diagnostic purposes - Laser fluorescence spectroscopy has been used for measuring fluorescence parameter of ocular media, pigments and also the exogenous fluorophores used for diagnosis or therapy. The technique of scanning laser ophthalmoscope used by Webb and his co-workers in 1980 to obtain fundus images and for exploring the clinical application in fluorescence angiography. It has advantages of having high spatial resolution, reduced light stress for the patient and good reproduction even when the pupil is small and media hazy.

Laser is light amplification by stimulated emission of radiation. The most useful property of laser light is concentration of light. Laser action has various parameters i.e. spot size (microns), exposure time (milliseconds, nano seconds and pico seconds) and power (milliwatt or milli joule). The exposure time determines the particular mode of action for laser modality.

Laser is essentially a destructive form of therapy depending on the absorption of light energy by ocular pigments and its conversion into heat energy. The mode of action for different lasers is as follows:

1.Photocoagulation e.g. Argon laser, Krypton laser, Dye laser, Diode laser.
2.Photodisruption e.g.YAG laser
3.Photoablation e.g. Excimer laser
4.Photoradiation e.g. Co_2 laser.

Tissue reactions observed depending upon type of lasers and their effects on biological tissue are, light toxicity, thermal effect, shock wave effect. Reparative effects or tissue reactions may be as:

a.***Photo coagulation:*** This is the thermal reaction of lasers which depends on the wave length of the laser, amount of energy, spot size and exposure time. This can be used for coagulation of blood vessels, formation of chorioretinal adhesion, and destruction of abnormal retinal pigment epithelial cells.

b.***Photodisruption:*** Laser infra red beam consisting of photons which when focussed force away electrons from the molecules producing plasma and shock waves which in turn disrupt the tissues. It acts mainly by mechanical disruption of tissues with ultra shot pulses.

c.***Photosensitization:*** Energy levels lower than the coagulation power induce a photochemical reaction. These reactions can be used therapeutically in ocular malignancies after sensitizing the tissue with hematoporphyrin derivatives i.e. in malignant melanoma. This can be done with Co_2 and dye lasers.

Principle of lasers depends on absorption of light energy of various pigments in ocular tissue i.e. hemoglobin, xanthophyll and melanin; and conversion into heat energy.

There are two techniques of laser delivery system:
1.Parafocal i.e. to produce smallest beam cross section on the retina.
2.Defocussing i.e. to produce change in size of

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Accepted: August 8,1995.

spots on retina.

Modes of delivery are:

a.Slit lamp
b.Indirect ophthalmoscope
c.Endophoto coagulation.

Treatment areas:

1.*Eye lids:* Lid tumors like hemangioma can effectively be treated by lasers.

2.*Cornea:* Laser photo coagulation to pterygium and blood vessels. Corneal sutures can be severed by lasers.

Excimer laser can be used for phototherapeutive and photo refractive purposes. High degree of myopia has been successfully corrected using excimer lasers.

3.*Ciliary body:* Trans scleral cyclophotocoagulation using ruby laser (red 694 nm) and neodynamic laser (1060 nm) have been found to be effective.

4.*Iris:* Laser iridotomy using ruby laser, argon laser and neodynamic laser makes an auxiliary opening in iris and relieves increased intraocular pressure, thus replacing surgical iridectomy. Photo mydriasis, can be effectively done with argon laser, when the pupil is too small and rigid. Traumatic iris cyst and a recurrent melanoma of iris can be shrunken with several sessions of argon laser.

5.*Trabecular mesh work:* Argon laser trabeculoplasty had dramatically changed the treatment of patients with uncontrolled glaucoma. Ruby laser can also be used for this purpose.

6.*Retina:* Retina is the main seat for lasers. Diabetic retinopathy, Eales disease etc. which previously led to vision threatening complications have been challenged by laser therapy resulting in prevention of severe visual loss and a stationary vision.

Treatment modalities in retina can be:

a.Panretinal photocoagulation. The goal is to destroy hypoxic or ischemic retina to remove stimulus for neovascularization and to allow better diffusion of oxygen from choroid.

b.Direct ablation of retinal or pre-retinal vessels: Heat is generated in retinal pigment epithelium, which results in obliteration of neovascular proliferative disease.

Retinal neovascularization occurs in following conditions:

1.*Vascular disease with ischemia:*

a.Diabetes mellitus
b.Eale's disease
c.Branch vein occlusion
d.Central retinal vein occlusion
e.Hypervis-cosity syndromes
f. Sickling hemoglobino-pathies
g. Retinopathy of prematurity

2.*Inflammatory disease with possible ischemia:*

a.Sarcoidosis
b.Retinal vasculitis
c.Uveitis
d.Pars planitis
e.Acute retinal necrosis.

3.*Direct ablation of subretinal neovascular membranes:*

Heat is generated in retinal pigment epithelium and in superficial choroid to ablate subretinal neovascular membranes.

4.*Elimination of diseased retinal pigment epithelium in:*

a.Central serious retinopathy
b.Retinal pigment epithelial detachment
c.Retinoschisis

5.*Destruction of neoplasms* by causing tissue necrosis e.g. retinal tumors (retinoblastoma) or choroidal tumors (e.g malignant melanoma and choroidal hemangioma).

6.*Production of chorioretinal adhesions* in retinal breaks, and peripheral retinal degeneration.

Complications: Complications of laser treatment are neglegible and can be managed without any permanent defect if recognized early. Temporary effects such as blurring of vision, corneal complications and pupillary abnormalities are result of thermal effects and are self limiting.

*Adverse effects include:*Increased intraocular pressure, iritis, pupillary distortion, lens opacities, hyphema, vitreous hemorrhage, foveal burn, optic neuritis, retinal holes and detachment. The beneficial effects are more than the harmful side effects and it should be explained to the patients.

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Case Report

Wheezing, Sinusitis, Wrist Drop in a 38 year old Asian Female

*Abdul Qadir Haji**, Mohammad M Siddiqui*, Tanveer P Mir**, Faroque A Khan****

Abstract: A 38-year-old Asian female presented with recurrent productive cough, wheezing, ethmoid sinusitis and a non-diagnostic bronchoscopic examination The patient subsequently developed mononeuritis multiplex with peripheral eosinophilia, chest infiltrates and antineutrophil cytoplasmic antibodies. Histopathology of open lung biopsy revealed necrotizing granulomatous vasculitis without eosinophilic infiltration. Both neurological involvement and eosinophilia responded to cyclophosphamide and corticosteroids and after six months of follow-up there is no renal involvement. This case represents a unique overlap between Churg-Strauss Syndrome and Wegener's granulomatosis. *JK Practitioner 1995; 2:184-8.*

Key Words: Asthma, Wegener's granulomatosis, Churg-Strauss syndrome, Antineutrophil cytoplasmic antibodies
Abbreviations: CSS- Churg-Strauss syndrome, ANCA- Antineutrophil cytoplasmic antibodies WG - Wegener's granulomatosis..

Introduction

The classification of vasculitis and a reappraisal of allergic granulomatosis and angiitis (Churg-Strauss Syndrome-CSS) was reviewed recently [1,2]. Wegener's granulomatosis may share some features with CSS but the two can be differentiated clearly [1,3-7]. While CSS is characterized by a strikingly uniform clinical picture of asthma, peripheral eosinophilia and eosinophilic infiltration of the involved tissues on histopathology, Wegener's granulomatosis (WG) is more likely to present with renal involvement and absence of asthma or peripheral eosinophilia [7-9]. Neurologic involvement particularly mononeuritis multiplex is classical of CSS but may occur in Wegener's granulomatosis as well [6-9].

Asthma is one of the major criteria in establishing a diagnosis of CSS [7]. Mean interval between the onset of asthma and the onset of vasculitis may range from 3 to 13 years [7], Since the mortality rates from asthma in United States nearly doubled from 1978 to 1987 [8,10] and there was an increase in asthma morbidity as well, CSS should be entertained in the differential diagnosis in any patient of asthma manifesting neurologic involvement, usually a peripheral neuropathy [6]. However, peripheral neuropathy has been reported in WG as well, although less frequently [11]. Similarly antineutrophil cytoplasmic antibodies (ANCA) are found in both CSS and WG [12,13].

We present a case which represents a distinct overlap between CSS and WG, with some atypical features.

Case report

A 38-year-old Asian female was admitted to the hospital with a two-month history of wheezing, productive cough and persistent low grade fever. She had not shown a favorable response to inhaled bronchodilators and prednisone. The cough was progressively worse with occasional blood streaked sputum. Ten months prior to the present admission, the patient had bilateral myringotomy for serous otitis media. On examination she was well nourished and in no acute distress except during the episodes of wheezing. Temperature was 37.8°C, blood pressure was 130/80 mmHg, pulse 98 beats per minute and a respiratory rate ranging from 20-24 per minute. Chest examination revealed bilateral scattered rhonchi. Rest of the physical examination was unremarkable. The CBC showed a white blood cell count of 9.7×10^9/l and hematocrit value of 35.1. Erythrocyte sedimentation rate was 91 mm/h. Serum creatinine and urinalysis values were normal. Chest X-ray film was normal and bronchoscopy non-diagnostic. Echocardiogram was essentially normal. CT scan of paranasal sinuses revealed ethmoid sinusitis (Fig. 1). Pulmonary function studies revealed a moderate obstructive ventilatory impairment with no significant bronchodilator response. While the patient was breathing room air, the partial pressure of oxygen

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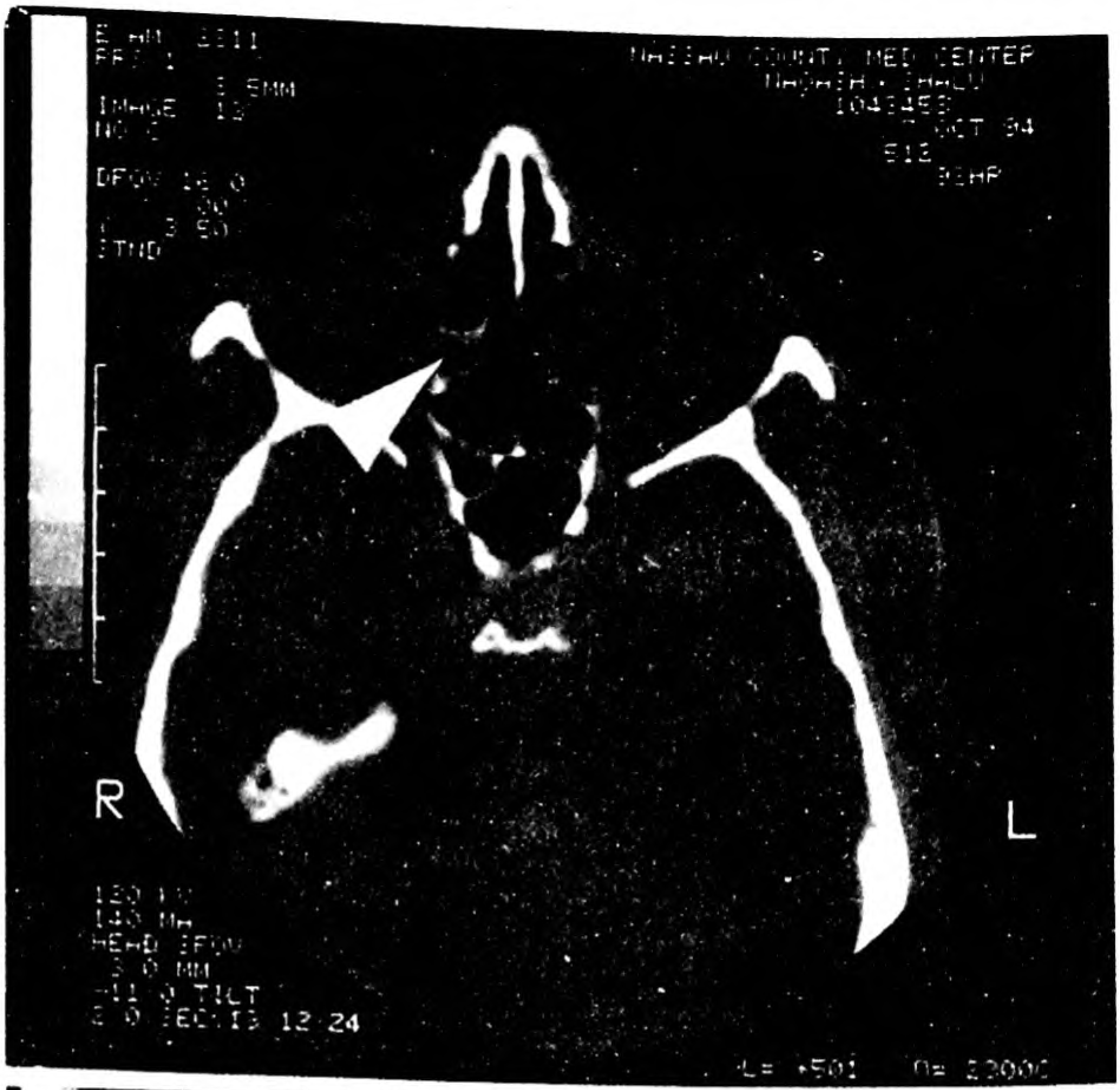
(paO_2) was 92.50 mmHg., the partial pressure of carbon dioxide ($paCo_2$) 40.20 mmHg and the pH 7.43. The respiratory flow volume loop was normal and a short trial of albuterol had no clinical effect. An otorhinolaryngologic examination revealed bilateral dry ears with myringotomy tubes in place and evidence of allergic rhinitis with postnasal discharge. The patient was a non-smoker housewife and her travel history included a stay in the Persian Gulf for seven years and a recent visit to India. There was no significant past or family history and she had no known allergies.

The patient was readmitted approximately two months after the first admission with sudden and severe sharp pain involving the left arm associated with numbness and weakness of left hand followed by numbness and pain in the right hand.

Physical examination revealed an ill-looking lady not in any distress. She was afebrile with a respiratory rate of 20 per minute and a BP of 130/80 mmHg. Examination of the chest revealed a few post tussive crackles in the left infrascapular area. There was no wheezing and no signs of congestive heart failure. Mentation and cranial nerves were normal but within next 24 hours patient developed left wrist drop.



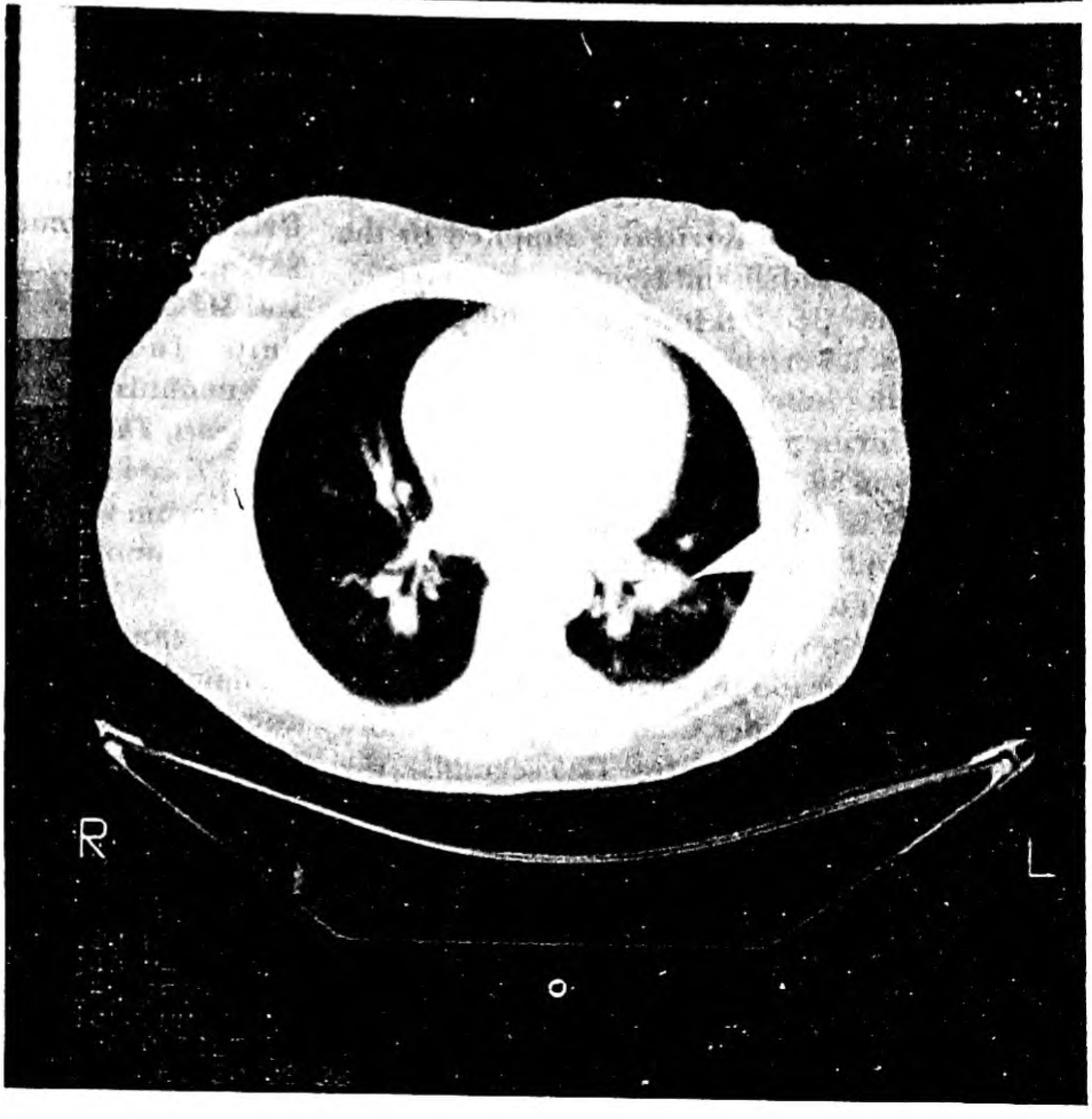


Fig.1.(top) CT Paranasal Sinuses showing ethmoid sinusitis.

Fig.2. (bottom)CT chest showing perihilar nodules and adenopathy.

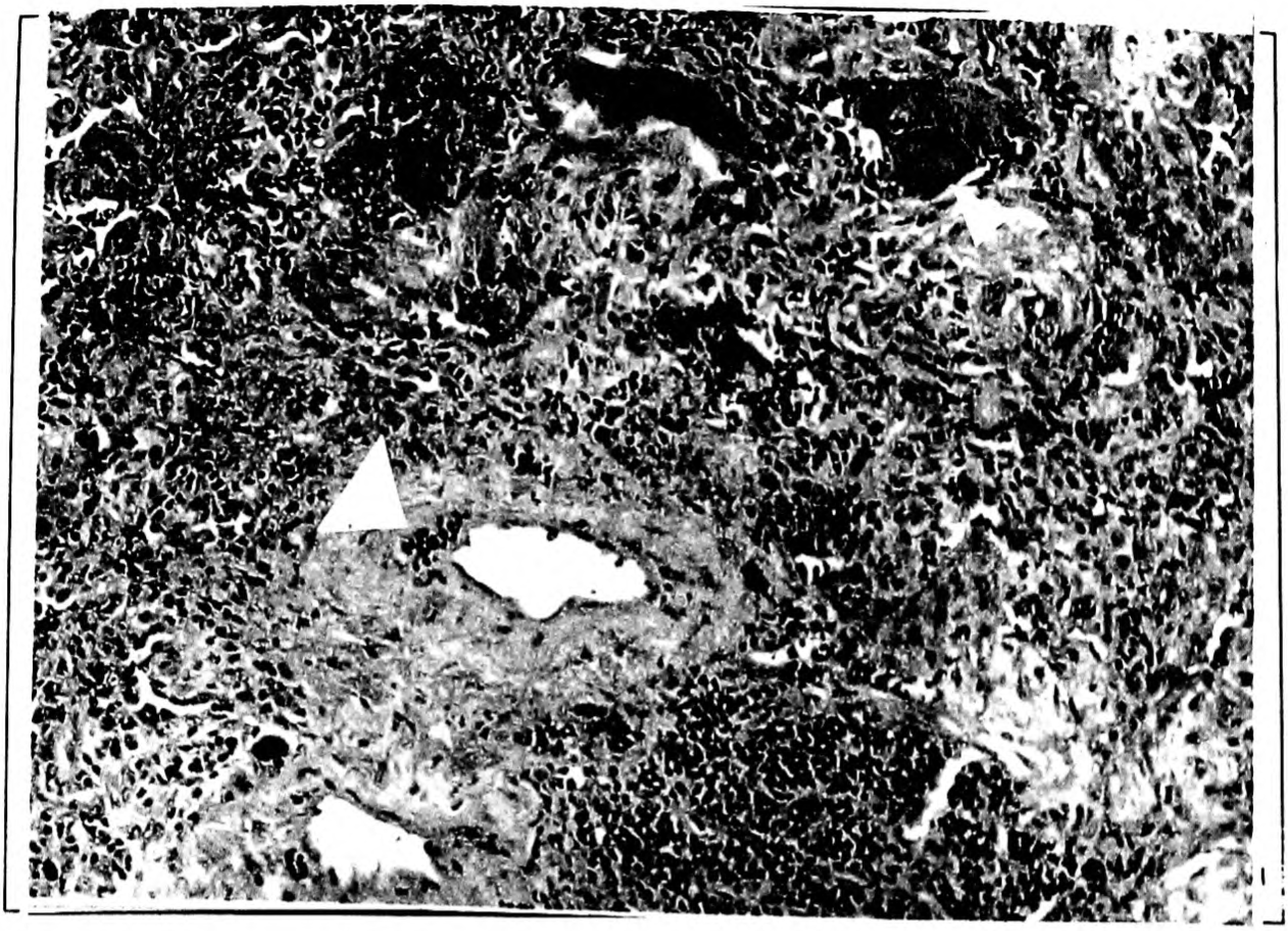


Fig.3. Dense inflammatory cell infiltrate permeating ping all layers of a pulmonary artery wall (large arrow). A multinucleated giant cell is prominent within the dense extravascular infiltrate (small arrow). Note the paucity of eosinophils. H&EX180

Neurological evaluation revealed sensory and motor deficits in the territories supplied by the left median, left radial and right ulnar nerves.

Chest X-ray revealed left lower lobe and perihilar infiltrates which were better seen on a lateral projection, and the tuberculin skin test response was 15 mm. The urine examination was normal. The hematocrit was 30.2 percent; white cell count on admission was 15,000 per mm^3 with 9% eosinophils (absolute eosinophil count of 1350) which gradually came down to 2% (absolute eosinophil count of 272) on the tenth hospital day. The platelet count was 150,000 and the erythrocyte sedimentation rate was 110mm per hour. The prothrombin time was 11.6 seconds (control 12.5 seconds); the partial thromboplastin time was 28.8 seconds. The angiotensin converting enzyme was 23.5U per liter (normal 8 to 52). The concentration of IgG was 1098mg, IgA 268mg and IgM 180mg per 100 ml; the concentration of IgE was less than 28IU perml (normal less than 103). The total hemolytic component (CH50) was 181 U per milliliter; C3 was 163mg, and C4 64.2 mg per 100 ml. Tests for anti-nuclear antibodies, rheumatoid factor, anti-native DNA, anticentromere, anti-RNP, anti-SSA and anti-SSB antibodies were negative. A repeated test for anti-MPO (P-ANCA) was positive measuring 1492 units (normal 0-10). A hypersensitivity pneumonitis screening for antibodies to *Aspergillus fumigates, Thermoactinomyces sacchari, Micropolyspora faeni, T. candidus, T. vulgaris, T. viridus* and to pigeon serum was negative. Microscopical examination of a stool specimen disclosed no ova or parasites.

A CT scan of the chest (Fig. 2) showed left perihilar nodules, left pleural thickening and mild adenopathy. Nerve conduction and electromyographic analysis were consistent with the clinical diagnosis of mononeuritis multiplex involving left median, left radial and right ulnar nerves.

On the 4th hospital day the patient underwent a left exploratory thoracotomy and microscopical examination of the lung biopsy revealed necrotizing and granulomatous vasculitis involving both the elastic and muscular arteries as well as veins of medium to small size; predominantly in peribronchial distribution. Special stains for acid fast bacilli and fungi were negative (Fig. 3).

The patient was started on prednisone and cyclophosphamide and showed both subjective and objective clinical response. After 20 weeks of treatment, the follow-up showed that the wrist drop had significantly improved and there was no peripheral eosinophilia or wheezing.

Discussion

In 1951, Churg and Strauss [4] reported 13 cases of severe asthma with a uniform clinical picture, including fever, hypereosinophilia and evidence of vascular disturbances in other organ systems. Pathological examination [4,5] revealed tissue infiltration by eosinophils, necrotizing vasculitis, and extravascular granulomas. WG, on the other hand, does not have asthma or tissue infiltration by eosinophils [7] but microhematuria (> 5 red blood cells per high power field) or red cell casts in urine sediment is included as a criterion for classification. From a clinical standpoint, patients with CSS have a classical triphasic clinical presentation [9]. Typically it begins with allergic rhinitis, which is often complicated by nasal polyposis and sinusitis; asthma and peripheral blood eosinophilia ensue, often accompanied by pulmonary infiltrates; and finally a systemic vasculitis develops that involves multiple organs, including the heart, skin and peripheral nerves; and renal involvement, when present, is generally mild [9]. The case under discussion represents a unique overlap between CSS and WG - while as wheezing and peripheral eosinophilia are in favor of CSS, pathological examination showing absence of eosinophil infiltration is in favor of WG (Table 1).

Feature	CSS	WG	Patient Under Discussion
Asthma	+++	Uncommon	Intermittent Wheezes
Eosinophilia	+++ (>10%)	Occasional modest	9% (on steroids)
History of Atopy	+++	Uncommon	Present
Upper airway destruction, deformity	Uncommon	+	Absent
Pulmonary nodules	Occasional nonfixed	++ fixed	++ fixed
Renal failure	+	++	Absent
Mononeuritis multiplex	+++	+	Present
C-ANCA*	Rare	90% of pts. during active disease	Absent
P-ANCA*	≤ 60%	1%-5%	Present

*Coarse, diffuse staining of ethanol-fixed neutrophils by antineutrophil cytoplasmic antibodies (ANCA)

**Perinuclear staining of ethanol-fixed neutrophils by antineutrophil cytoplasmic antibodies.

Table-1.Churg Strauss Syndrome (CSS) and Wegener's Granulomatosis [7,8,15]. Comparison & Contrast with the Case Under Discussion.

Transient wheezing rather than a uniform clinical picture of asthma is an atypical feature in this case. Recently criteria for the classification of Churg-Strauss syndrome were developed [8]. Six features were selected - asthma, the presence of more than 10 percent eosinophils in the blood, mononeuropathy including multiplex or polyneuropathy, non-fixed pulmonary infiltrates demonstrable on X-ray films of the chest, a paranasal sinus abnormality, and biopsy specimens containing a blood vessel with extravascular eosinophils. The detection of four or more of these criteria had a sensitivity of 85 percent and a specificity of 99.7 percent. Our patient had wheezing, mononeuritis multiplex and paranasal sinus abnormality. She also had peripheral eosinophilia (9 percent) while on low-dose steroids. This eosinophilia gradually subsided after starting cyclophosphamide and a higher dose of steroids. She has had no evidence of renal involvement during a follow-up period of six months. During the second admission, patient also developed detectable anti-neutrophil antibodies against myeloperoxidase. This particular antibody is over 99 percent specific for Wegener's granulomatosis, microscopic polyarteritis nodosa and primary necrotizing and Crescentic glomerulonephritis [12]. Other ANCA associated vasculitides include CSS and the classic polyarteritis nodosa [13]. In fact presence of ANCA is not included as a criterion for either CSS or WG [7,8]. However, the late development of this antibody in our case was rather atypical.

It was interesting to note in this patient that mononeuritis multiplex and eosinophilia both appeared at the same time and as the neurologic syndrome started improving, eosinophilia also showed a downward trend. Hypereosinophilia and mononeuritis multiplex and hallmarks of CSS

[14], but a transient hypereosinophilia during the early stage of mononeuritis multiplex is not reported and may represent an undefined manifestation of an indeterminate pathogenetic mechanism.

CSS and Wegener's granulomatosis can present as a distinct overlapping clinicopathologic syndrome; transient hypereosinophilia could be a harbinger of neurological involvement and brief episodes of airways hyperresponsiveness rather than long standing asthma can be a manifestation of such an overlap syndrome.

Trivial wheezes masquerading as bronchial asthma and not responding to the conventional treatment may be a subtle manifestation of an underlying uncommon potentially lethal disorder if not recognized and treated early. This case exemplifies the importance of the statement "*all that wheezes is not asthma*".

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Case Report

5 Fluorouracil Induced Bradycardia - Is it Autonomic Imbalance?

Shiekh Aejaz Aziz, Khalid Mohiuddin, Mushtaq Ahmad, Ajaz Lone, Gul Mohammad Bhat, Hajra Bano.

Abstract: Occurrence of sinus bradycardia in a young female, after prolonged use of 5-Fluorouracil. Vagolytic test (Atropine test) was done, and sinus bradycardia attributed to a hypervagotonic state induced by 5-Fluorouracil. *JK Practitioner 1995; 2: 189–91.*

Key words: 5-Fluorouracil, Sinus bradycardia

Case Report

AQ, 28 years young female, presented last year with features of subacute intestinal obstruction. She was evaluated and found to have a strictural lesion of the splenic flexure of colon. She was operated upon and histopathological examination of the resected specimen revealed adenocarcinoma $T_2N_2M_0$. Patient received adjuvant chemotherapy in the form of Inj. 5-Fluorouracil 1.5 g/m^2 CIV-24 hours (day I) and Levamisole 150 mg/m^2 (day 1,2 and 3) PO, each such cycle of chemotherapy to be repeated every 21 days. Pre-chemotherapy chest X-ray and ECG were normal (Fig. 1). After the elapsing of 7 1/2 months from the first cycle of chemotherapy, patient reported with complaints of distension of abdomen. Clinical examination revealed her to be in PS I (ECOG), pulse rate of 48/minute, regular, BP 120/80mmHg, with normal examination of her chest, and cardiovascular system was normal. Abdominal examination revealed ascites. Fundus-occuli were normal. Laboratory investigations revealed Hb 12.4g%, serum bilirubin 0.6 mg% (normal upto 1.4 mg%), serum alkaline phosphatase 279 U/l (normal upto 300 U/l). Serum sodium was 135meq/l, potassium 4meq/l, calcium 10mg/dl, magnesium 2.5 mg/l. Serum T4 levels were 7ug/l (normal 5-12 ug/l), T3 134ug/l (Normal 70-190 ug/dl), TSH 3 miU/l (normal less than 5 miu/l). ECG revealed sinus bradycardia 48/minute, regular rhythm. Echocardiography (M mode and real-time) was normal. Patient was subjected to provocative test for a possible structural nodal disease, however, when 2 mgms of Atropine sulfate were given intravenously, the sinus rate increased to 107 beats/minute (figure 3). The findings registered in the first cycle of chemotherapy were re-documented during the 2nd and 3rd cycle of chemotherapy.

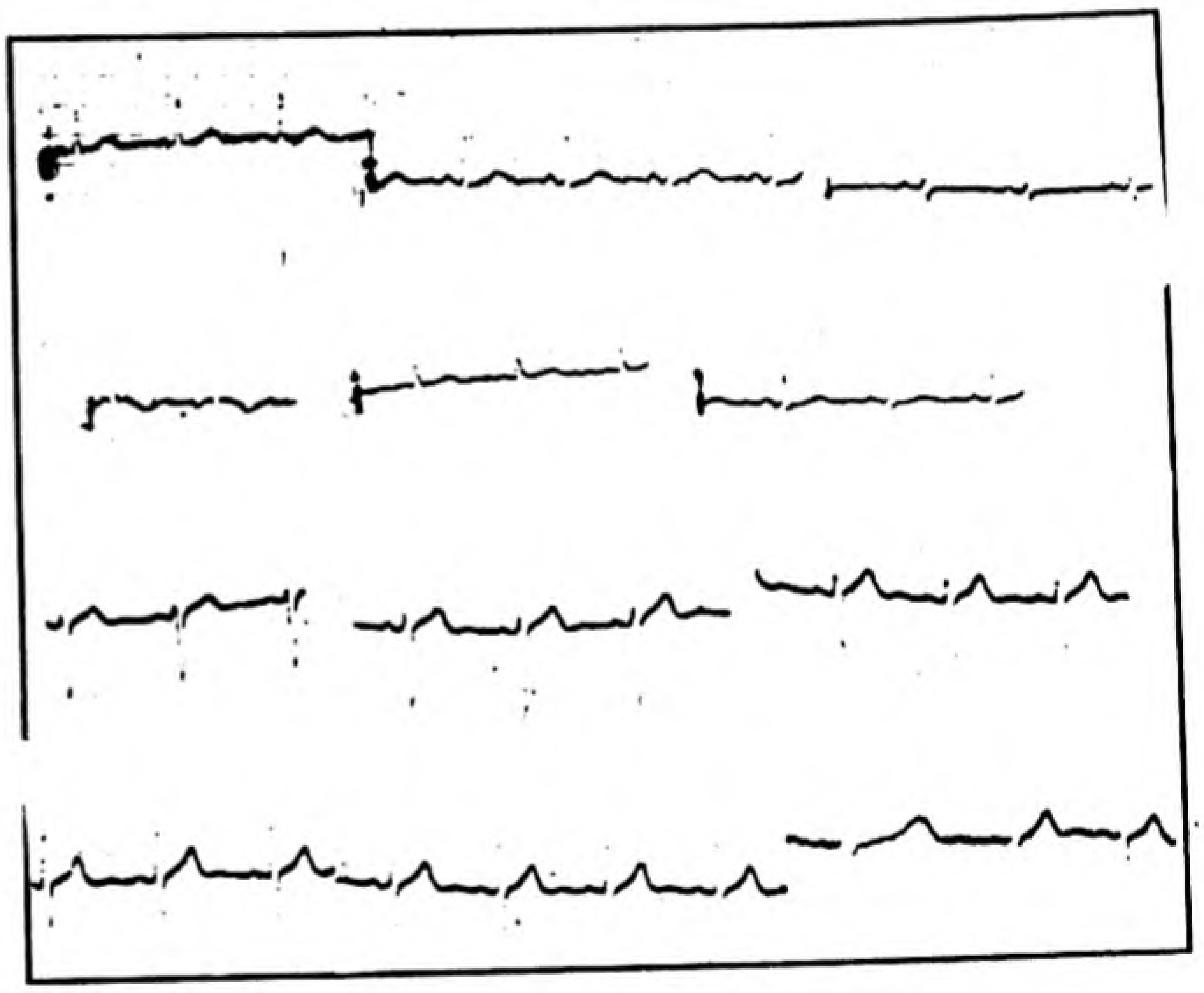
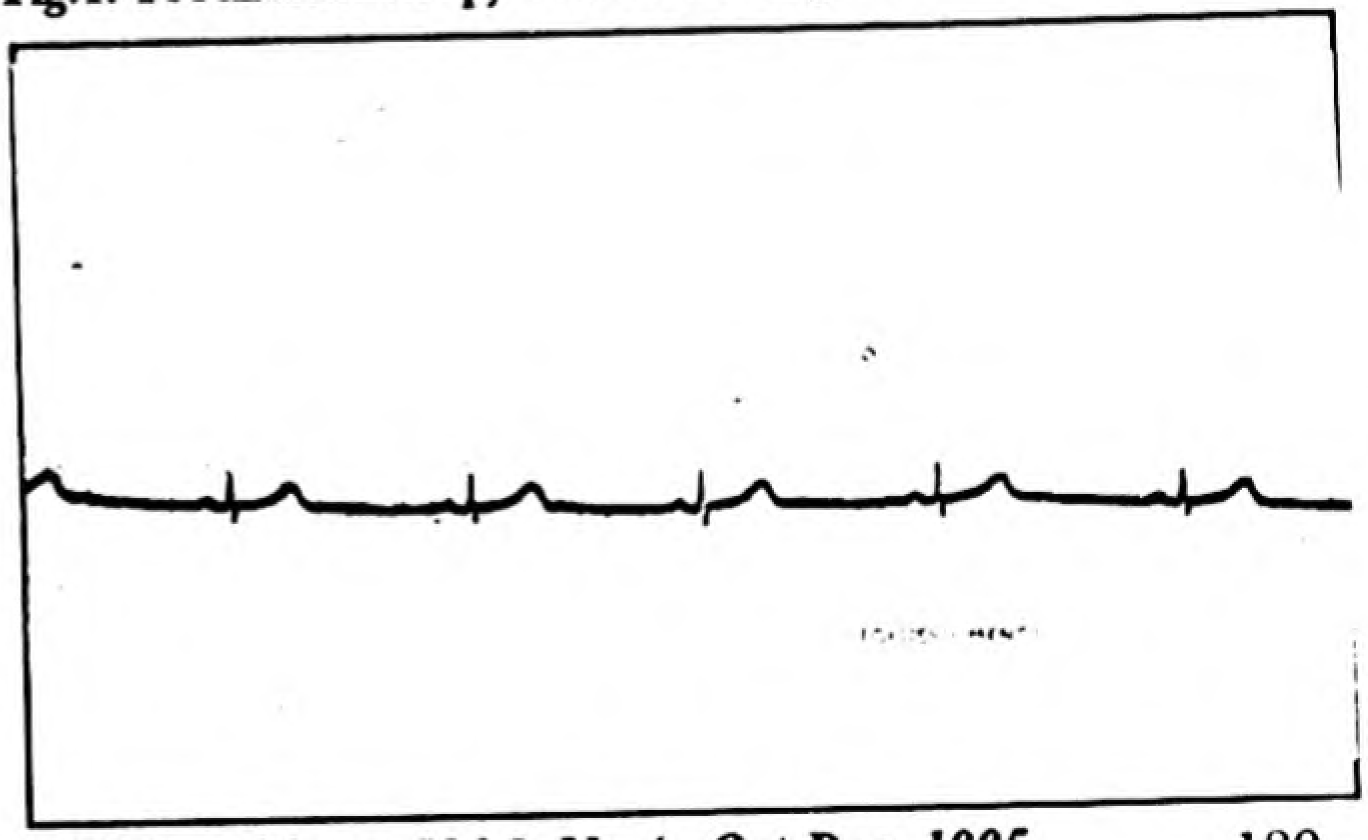


Fig.1. Prechemotherapy electrocardiogarm



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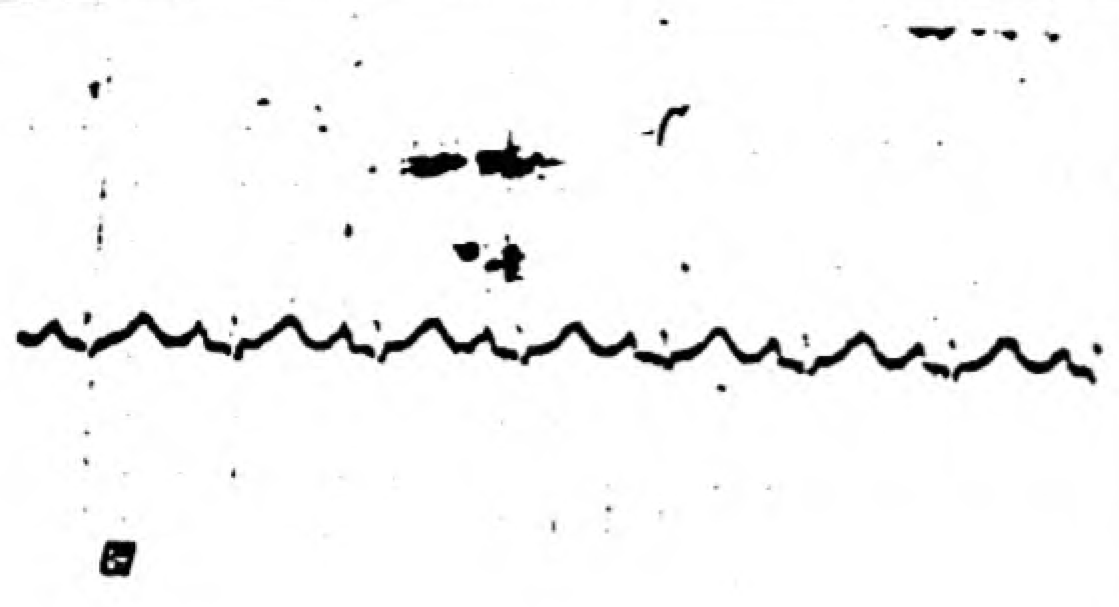

FIG.NO.3. SINUS TACHYCARDIA

Discussion

5-Fluorouracil is a semi-synthetic, S phase-specific-pyrimidine antimeta-bolite, active against various malignancies. In 1975, Dent and McColl pioneered to report that this drug has potential of clinical cardiotoxicity [1]. Further studies on this topic revealed an incidence of cardiotoxicity to the tune of 1.6% in retrospective studies [2], while in prospective studies, it is higher (including our, unpublished data). Keeping in view the increase in sinus rate from 48/minute to 107/minute after 2 mg of atropine sulfate i/v, we conclude that sinus bradycardia in this patient was a manifestation of a hypervagotonic state. Since bradycardia was temporally associated with the use of 5-Fluorouracil, we assume that the association is causal. The same is substantiated by excluding other causes of bradycardia after clinical examination and various investigations.

The etiopathogenesis of the cardiotoxicity of this drug needs to be defined precisely [3]. However, autoimmunity, direct toxicity due to metabolite FAC (Fluoroacetaldehyde), vasospasm, increased thrombogenecity, and stunned myocardium (post-ischaemic left ventricular mechanical dysfunction) are presumed to be possible mechanisms [4-7].

The cardiotoxicity manifests itself as chest pain, rise in cardiac enzymes, angina pectoris, Prinzmetal angina, myocardial infarction, cardiogenic shock (sometimes reversible), sudden death, hypotension, repolarisation abnormalities, sinus tachycardia, atrial pre-mature beats, atrial fibrillation, ventricular premature contractions and ventricular tachycardia [8].

Though anthracycline induced autonomic dys function has been reported (in addition to th cardiotoxicity), autonomic imbalance particularl 5-Fluorouracil induced hypervagotonic state has not been described so far in the world literature in the armamentarium of 5-fluorouracil cardiotoxicity. This statement is substantiated by the fact that while assessing the autonomic dysfunction due to anthracyclines in a recently conducted study, the patients receiving 5-Fluorouracil based chemotherapy were kept as controls.

Further vigilant and careful studies need to be carried out to substantiate our observation.

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Case Report

Disengaged Threaded Steinmann's Pin Presenting as Parietal Wall Sweling

Nisar Ahmad Wani, Mumtazudin Wani, Gh. Hassan Bhat, Dr. Mohd Shafi Malik.

Abstract: A 50 year old male presented with a parietal wall abscess in the left upper abdomen. Upon surgical exploration granulomatous reaction around a Steinmann's pin was found, which had migrated from the site of an earlier shoulder fixation. *JK Practitioner 1995;2:191-2.*

Key words: Abscess, Parietal wall, Abdomen

Introduction

It has been well documented in literature that migration of foreign implants used for internal fixation of fractures in orthopedic surgery can occur from its place of insertion to another place [1], thereby either presenting as a swelling somewhere else or resulting in visceral injury to nearby organs. We are also emphasizing and presenting a similar case in which migration of Steinmann pins used for internal fixation for fracture of head of left humerus had occurred and presented as a subcutaneous swelling in the left hypochondrium of abdomen.

Case Report:

A fifty year old male was admitted in the surgical ward of Medical College Hospital Srinagar, with the complaints of pain and swelling in the left upper abdomen of one month duration. There was no history suggestive of any systemic involvement or any abdominal trauma. Patient gave history of vehicular accident six months back, when he suffered fracture of head of left humerus and at that time internal fixation of fracture was done by means of Steinmann pin.

Examination revealed an average built male having a 2.5x8.0cm parietal wall swelling in left hypochondrium. The swelling was firm, tender, oblong with regular margins without any adherence to skin or underlying structures. Rest of the physical and systemic examination was normal.

Patient was given a course of antibiotics and then subjected to exploration which revealed the presence of Steinmann pin with granulomatous reaction around it (Fig. 1). The pin was removed and after ensuring proper hemostasis, the wound was closed. In the subsequent post operative period x-ray of left shoulder revealed a single Steinmann pin, the other one having migrated subcutaneously to the site of swelling. The patient was asymptomatic at discharge.

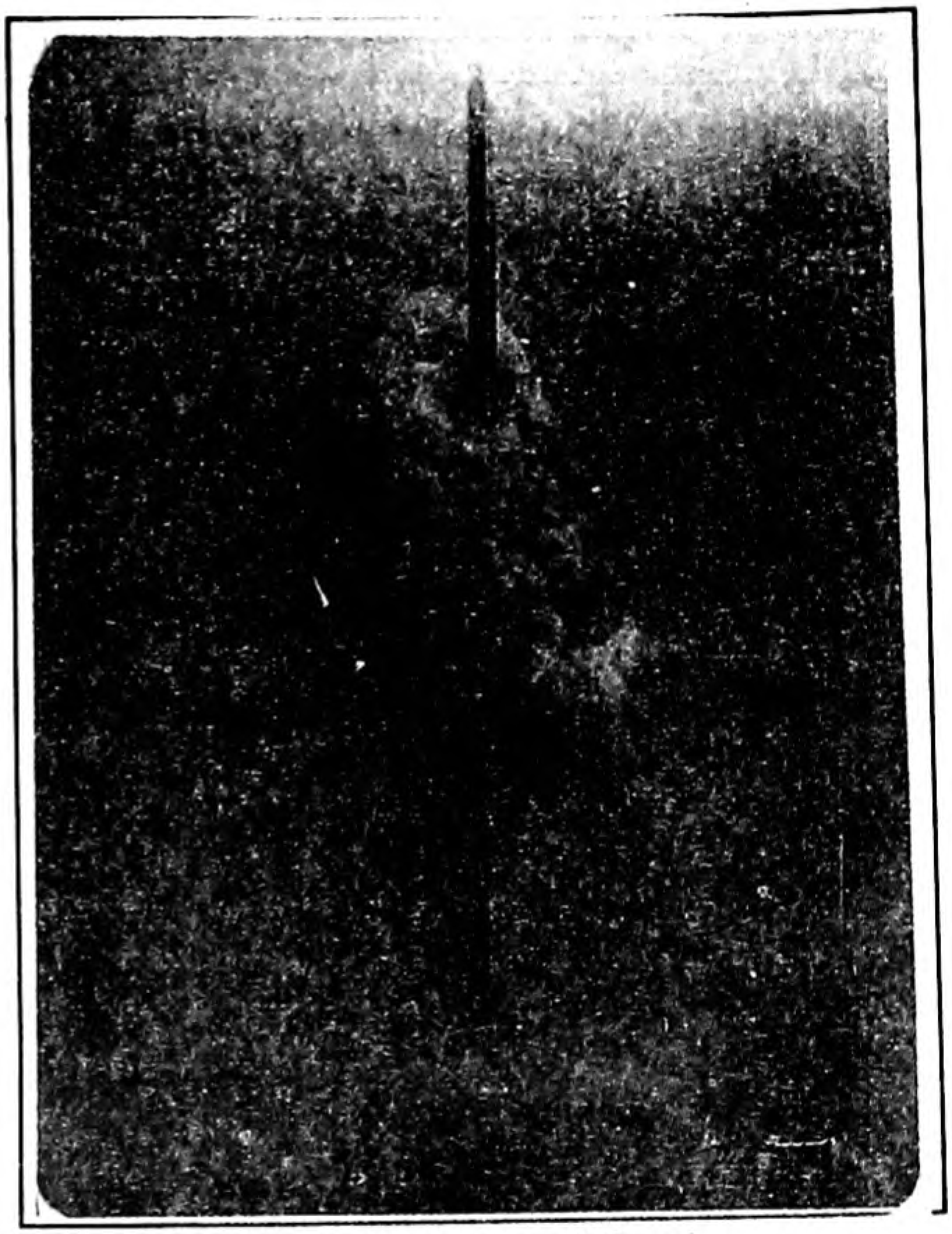

Fig.1. Granulomatous mass around the pin.

Discussion

Foreign implants like threaded pins, screw plates and nails used for internal fixation in orthopedic surgery are likely to migrate from their place of insertion to other sites [1]. The risk is increased by the motion at the fracture site which can cause loosening, breakage or disengagement of the fixa-

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tion device. Brodel and Leve [2], described a case of intrapelvic protrusion of a hip screw. Perforation of bladder [3], rectum [4], and injury to common iliac vessels [5] have been reported after fixation of hip joint fractures. Similarly in our case migration of Steinmann pin had occurred from left shoulder to left hypochondrium which emphasizes the fact that such migration can take place aided by mobility of a particular joint. The appearance of a swelling following any of these fixation procedures, albeit after long periods, should prompt the surgeon to think of a possibility of the swelling being related to migration of the fixation device.

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Dr. G. M. Malik

Case Report

Paradoxical Expansion of Cerebral Tuberculomas

G M Malik,[+] J A Basu,[*] M Mubarik,[•] T. Hussain, S. Rashid,[**] S. A. Kadla.[*]

Abstract: A 28 year old female was diagnosed as having Pott's spine with paravertebral abscess. She was advised a plaster jacket and complete bed rest in addition to antitubercular treatment (ATT). The patient, while showing a marked improvement of her Pott's disease on ATT, developed (R) sided focal seizure with secondary generalization and raised intracranial tension on two separate occasions, which responded each time to the addition of decongestant therapy and anticonvulsant therapy. CT Scan of the brain was suggestive of left cerebral tuberculomas with surrounding edema on each occasion. Paradoxical appearance of tuberculomas should be suspected and looked for, if a patient develops a neurological dysfunction of this sort while on a seemingly successful course of ATT for tuberculosis elsewhere in the body. *JK Practitioner 1995; 2: 193-6.*

Key words:Tuberculosis, Tuberculoma, Pott's spine.
Abbreviations: ATT-Antitubercular treatment, RIF-Rifampicin, INH-Isoniazid, PZA-Pyrazinamide, EMB-Ethambutol.

Introduction

The incidence of tuberculosis in the industrialized world has declined in recent years, but elsewhere, for example in India, the disease continues to be common [1,2]. Cerebral tuberculomas are the commonest extra-pulmonary lesion, presenting as a tumor like masses of characteristic granulomatous tissue which produces symptoms of an intracranial space occupying lesion [3]. Computed tomography (CT) has been a major advance in the diagnosis, management and monitoring of these tuberculomas [4,5]. Some earlier reports indicate the appearance or expansion of these tuberculomas during a successful course of treatment for tuberculosis elsewhere in the body [4,6]. The present paper concerns one such patient who, while on a successful course of antitubercular treatment for Pott's spine developed a paradoxical appearance or expansion of an already existent left cerebral tuberculomas. This is the first case of its kind reported from Kashmir valley which has a high prevalence of tuberculosis.

Case Report

A 28 year old female was diagnosed as a case of Pott's spine (thoracic 8 & 9 vertebral level) with paravertebral abscess. She has been prescribed antitubercular treatment-ATT (Isoniazid-300 mgs, Rifampicin 45- mgs, Ethambutol-800 mgs and Pyrazinamide-1.5 gms) and pyridoxine-20 mgs/

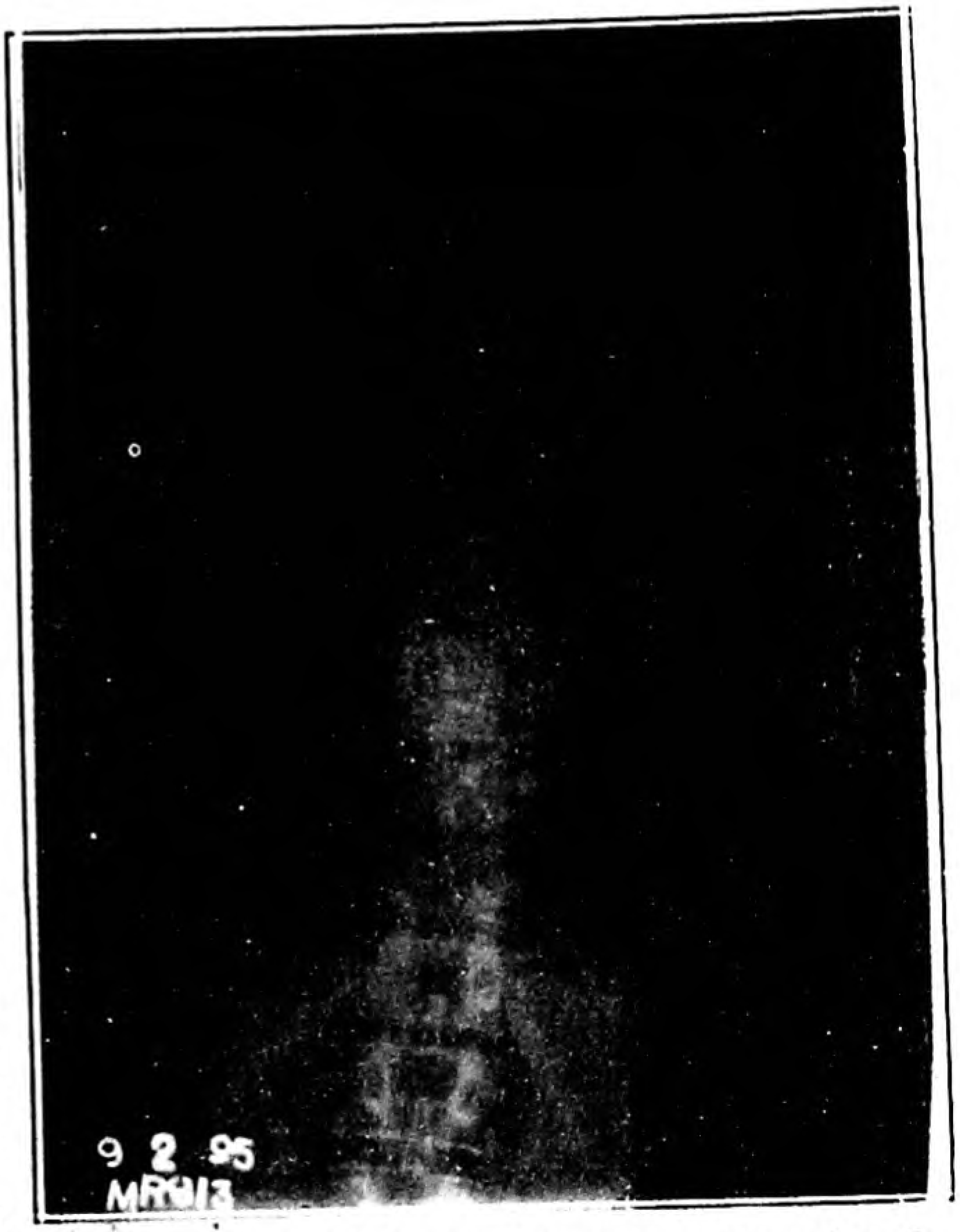

Fig.1. Radiograph dorsal spine (AP) showing destruction of T8 & T9 vertebrae & a paravertebral abscess

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once daily. Also, a plaster jacket and bed rest was advised. While on the above prescription, the patient felt significant improvement of her symptoms and became ambulatory with support within 35 days. But, after having completed 44 days of the treatment she developed global headache with vomiting, of increasing severity. She was hospitalized the next day and in hospital she developed a focal seizure in her right foot with secondary generalization. Post ictally she complained of weakness right half of the body, which started improving after 2 hours. Patient denied a history of head trauma. There was history of tuberculosis in a close family member,about 2 years back. She had not received any immunizations and was in lactational ammenorrhea.

Pertinent findings on physical examination included mild pallor, blood pressure of 160/104 and a plaster jacket applied round lower thoracic and lumbar spinal vertebrae. The neurological examination revealed bilateral papilledema, hypotonia with muscle power of grade 3/5 in right upper and lower limbs and bilateral upgoing plantars. The neurological examination on the second day revealed positive right sided grasp and palmo-mental reflexes, with positive glabellar tap, rest of the examination being normal. From sixth day onwards, right sided frontal lobe signs could not be elicited. Rest of the general physical and the systemic examinations were normal.

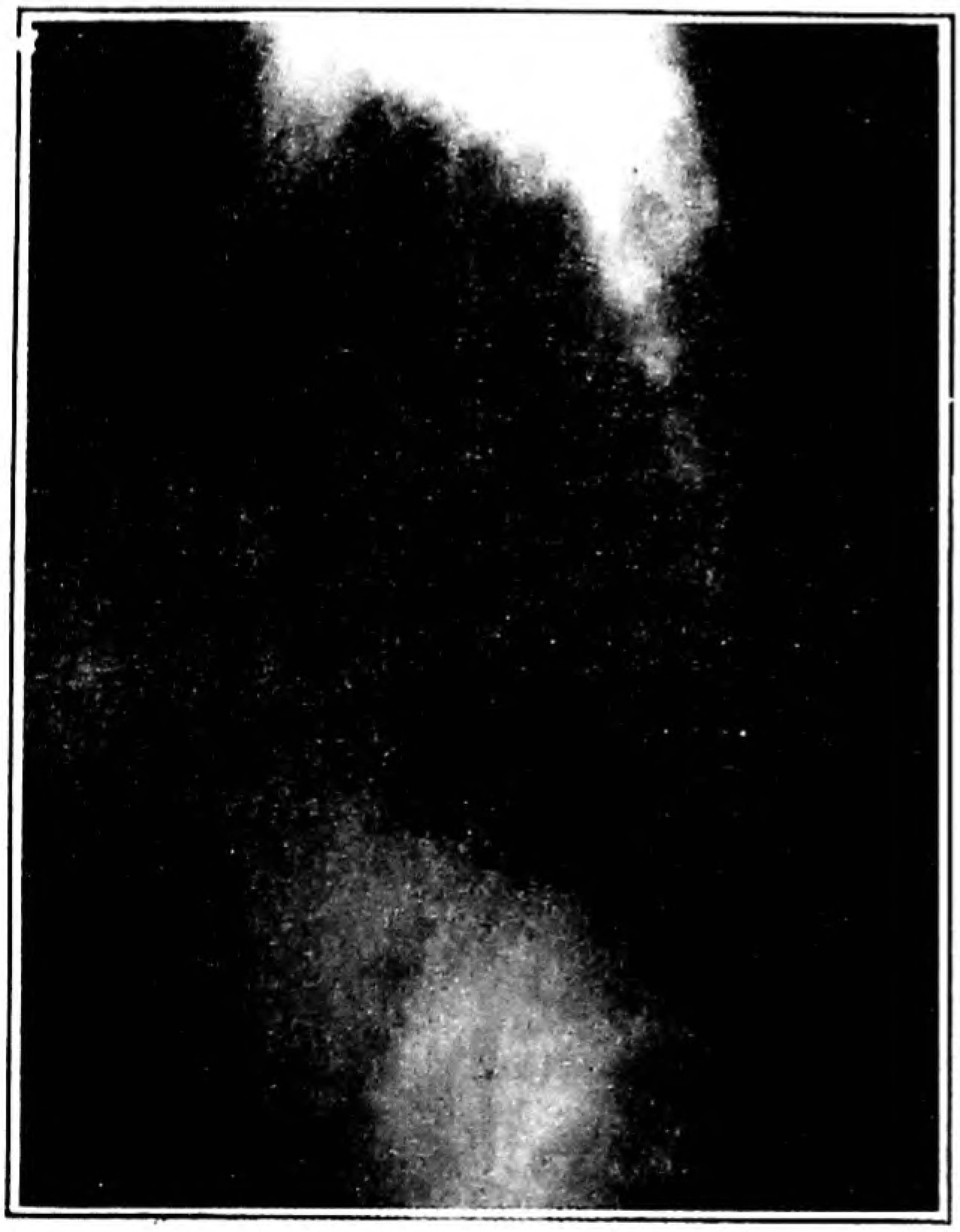
Fig.2.Radiograph of the dorsal spine (lat.) showing collapse D8 and D9 vertebrae

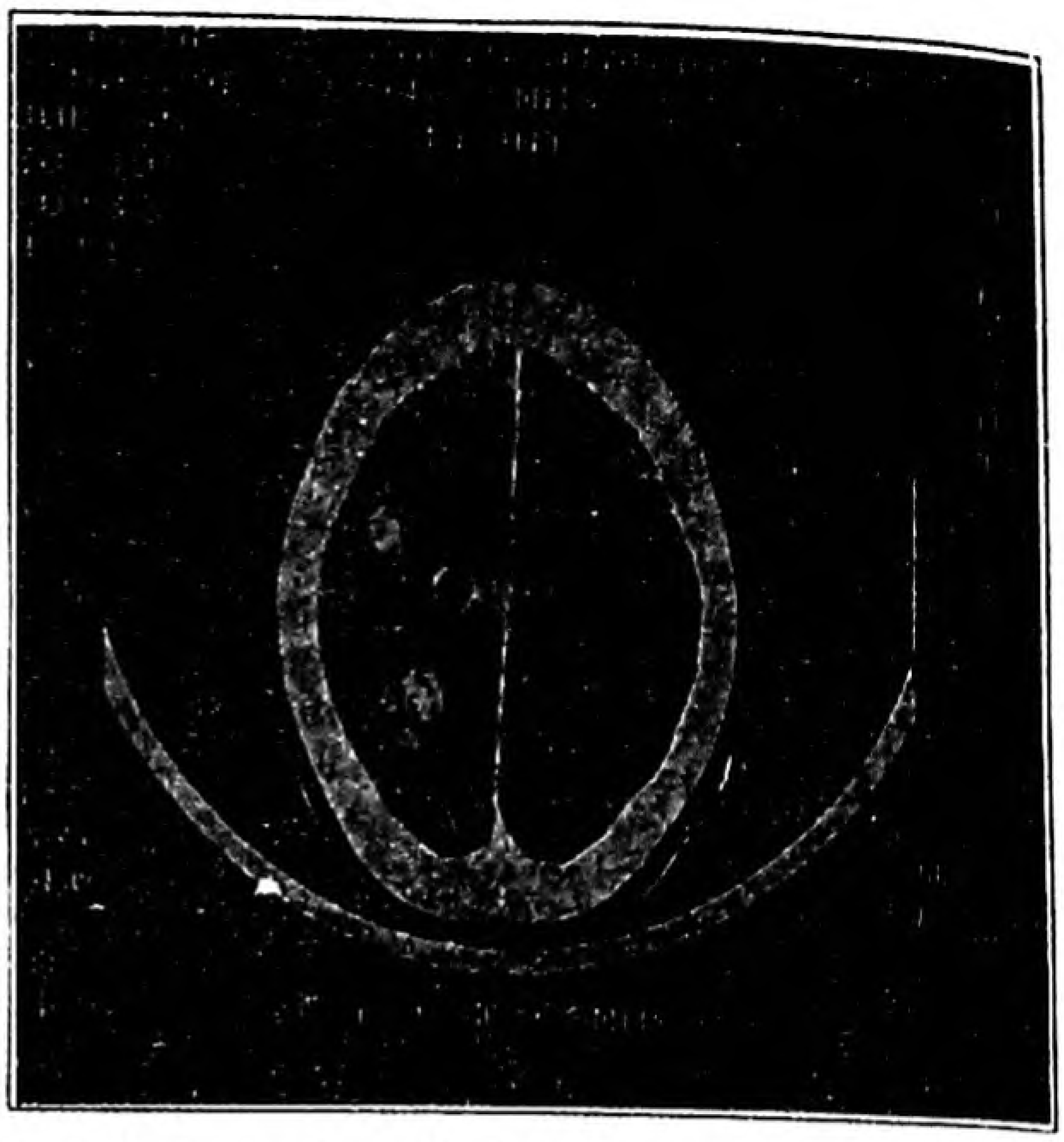
Fig. 3. Contrast enhanced CT showing multiple ring enhancing lesions in left cerebral hemisphere

The hemoglobin concentration was 9.0 g/dl. Radiographs of the spine revealed destruction of thoracic 8 & 9 vertebral bodies with collapse, loss of disc space and a paravertebral abscess (Fig.1). Contrast enhanced CT scan of skull revealed multiple hypodense areas in the left cerebral hemisphere with ring enhancement with surrounding edema, suggesting tuberculomas (Fig.2). Lumbar puncture was not possible in view of the plaster jacket. Rest of the haemogram, urine examination, blood chemistry, X-rays (Chest and skull) and ECG were normal. ELISA for HIV was negative.

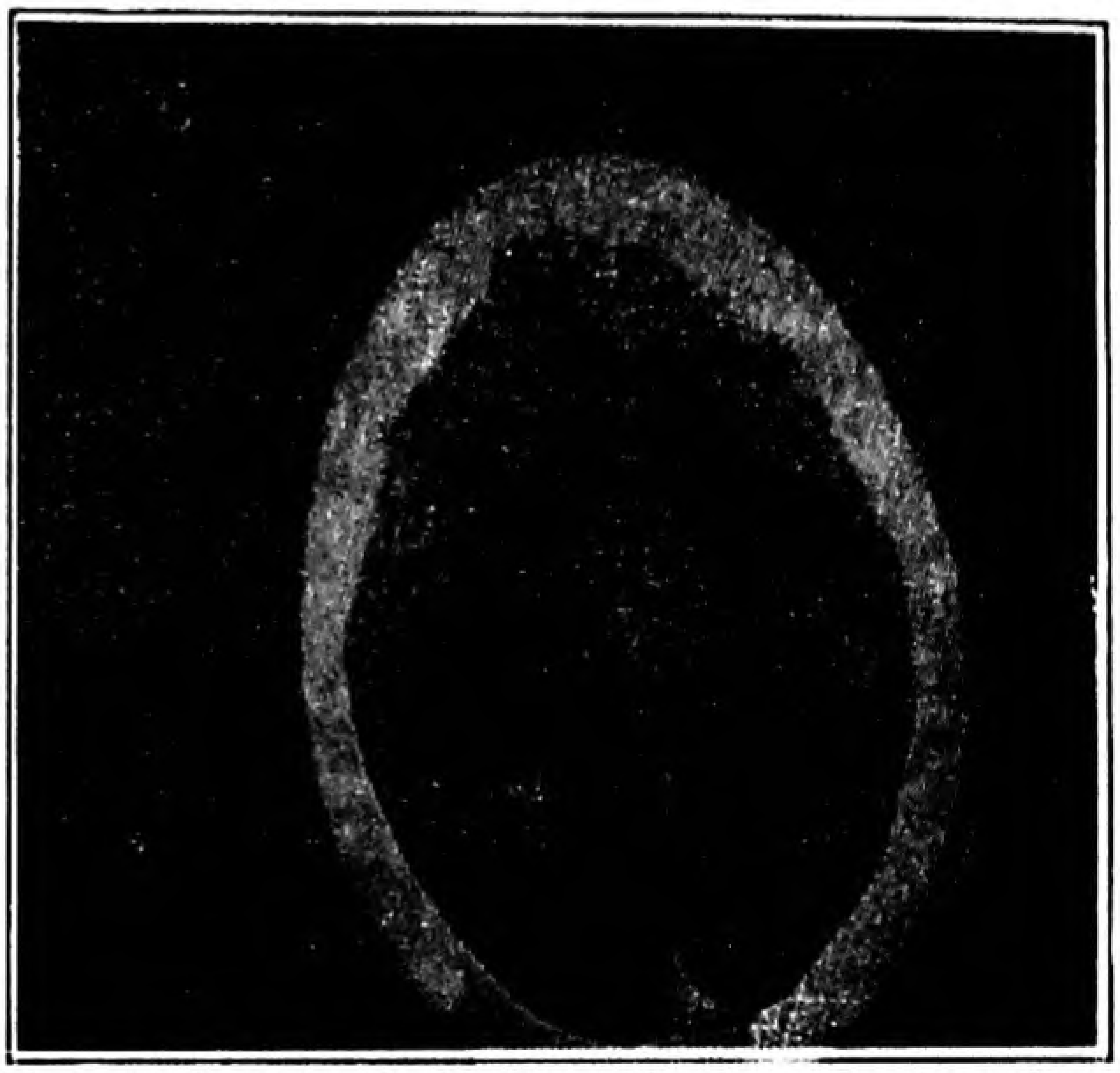
Fig.4. Persistent cerebral lesions with edema after 4 months

In addition to continuing ATT, the patient was managed with decongestant therapy in the form of mannitol (40 gms by vein over 30 min q 6 hourly - day 1), glycerol (30 gms q.i.d oral for 15 days) and dexamethasone (8 mg by vein tid for 10 days). After 10 days, prednisolone (60 mg daily, oral) was started instead of dexamethasone which was tapered over a period of 2 months. In addition she was given phenytoin and Nifedipine. PZA and EMB were stopped after 3 months. She improved clinically as evidenced on weekly follow up. Papilledema resolved completely over a period of 2 months. Four months after the first discharge, the patient landed again with a right focal seizure and raised intracranial tension. The repeat contrast CT scan revealed the persistence of these tuberculomas with surrounding edema (Fig.4). The reinstitution decongestant therapy again led to clinical improvement. The patient is continuing on ATT (RIF 450 mg + INH 300 mg, Pyridoxine 20 mg) and steroids (Prednisolone 20 mg).

Discussion

In India, where tuberculosis is widespread, tuberculomas constitute a significant percentage of intracranial space occupying lesions [3,5], being the commonest extra-pulmonary lesion and presenting usually with either focal epilepsy or with symptoms and signs of focal lesion or of raised intracranial tension [4]. Tuberculomas are known to occur in patients with past history of tuberculosis or evidence of tuberculosis outside the nervous system [5]. Further, some earlier reports indicate that tuberculomas can paradoxically appear, expand or proliferate while a patient is on a successful therapy for tuberculosis elsewhere in the body [4,6-10] and the same also holds true for the present patient.

Clinical experience has revealed a high prevalence of tuberculosis in any form in the Kashmir valley. Our patient, while showing a marked improvement of her Pott's disease on ATT, developed focal neurological sings and raised intracranial tension due to CT proven tuberculomas. Whether the tuberculomas developed afresh or there was expansion of already existing ones, could not be ascertained as the CT scan was not done before starting ATT. However, both possibilities are known to occur in these patients [11].

The cause (s) of sudden paradoxical appearance while the patient is responding to treatment for tuberculosis elsewhere, is not fully known. The response to treatment shows that such incidents cannot be attributed to irregular medication, drug resistance or failure of the drugs to penetrate the blood-brain barrier [12]. Some investigators believe that this reaction may be due to an immune response at the site of these lesions in the brain which instead of being discharged (as in the case with a pulmonary infection), cause neurological symptoms and signs [4,12,13].

The appearance or expansion of the tuberculomas has usually been observed when the patient was on treatment for about 3 months, but some have reported the response to occur as early as within 30 days and as late as 12 months [4, 11]. The paradoxical reactions of tuberculomas are known to occur repeatedly even after completing the treatment [5]. Our patient developed this paradoxical response on two separate occasions (i.e. after 44 days and after 4 months). The advent of CT scan has led to the increased recognition of these lesions which are usually seen as dense enhancing masses with surrounding edema [4]. The three groups of these lesions namely early, old and mature can be identified on contrast CT scan as showing uniform, ring or no enhancement respectively [14].

The clinical significance of tuberculomas appearing while a patient is on ATT lies in the fact that these can cause repeated episodes of raised intracranial tension with its attendant hazards. As a result, a patient even on an effective chemotherapy for tuberculosis elsewhere may succumb to raised intracranial pressure. The surgical intervention of these tuberculomas remains obscure on account of their multiplicity. The mainstay of treatment is an effective ATT with decongestant therapy as the eventual resolution may occur with medical treatment alone [4]. Further, in view of the recurrence of these paradoxical reactions, it is suggested that such patients should be kept under observation for many years until the efficacy of ATT has been assessed more rigorously.

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Short case report

JK Practitioner 1995;2:196-8.

Unusual Association of Neurofibromatosis with Chilaiditi's Syndrome

A.S. Sethi[*], *K. Ahmad*[**], *Aleem Jan*[***]

Neurofibromatosis (Von Recklinghausen disease) is an autosomal dominant disorder affecting 1 in 4000 persons and approximately fifty per cent of cases representing new mutations [1]. The disorder may affect any organ system of the body. We describe a case of neurofibromatosis associated with Chilaiditi's syndrome, who also had abdominal tuberculosis.

Case report:

A 12 year old male child, lone issue of his parents was admitted with complaints of progressive distension of abdomen, colicky abdominal pain, radiating to right shoulder, vomiting, episodes of diarrhea alternating with constipation and evening rise of temperature. Distension of abdomen had become more pronounced from last five months, whereas abdominal pain and vomiting had been a constant complaint for last four years. There was a positive family history of tuberculosis.

Physical examination of the boy revealed pallor and signs of multiple vitamin deficiencies. There were multiple cafe-au-lait spots over front and back and multiple sessile nodular swellings (Figure 1). There were no peculiar facies, icterus, thyromegaly or lymphadenopathy. The height was 130 cms, head circumference 55 cms, weight 28 kgs, B.P. 110/70mm Hg. There was no B.C.G scar, examination of chest and cardiovascular system revealed no abnormality. On abdominal examination, fluid thrill and shifting dullness were elicitable. Liver was palpable 4 cms below right costal margin, soft, non tender with rounded margins and spleen was palpable 2 cms. below left costal margin. Higher functions were normal and there was no cranial nerve palsy. There was wasting of left thenar and hypothenar eminences. Motor and sensory system were normal. There was no cerebellar dysfunction and no sign of meningeal irritation. There was bowing of left leg. Rest of the musculoskeletal system examination did not reveal any obvious abnormality. Slit lamp examination of eyes confirmed

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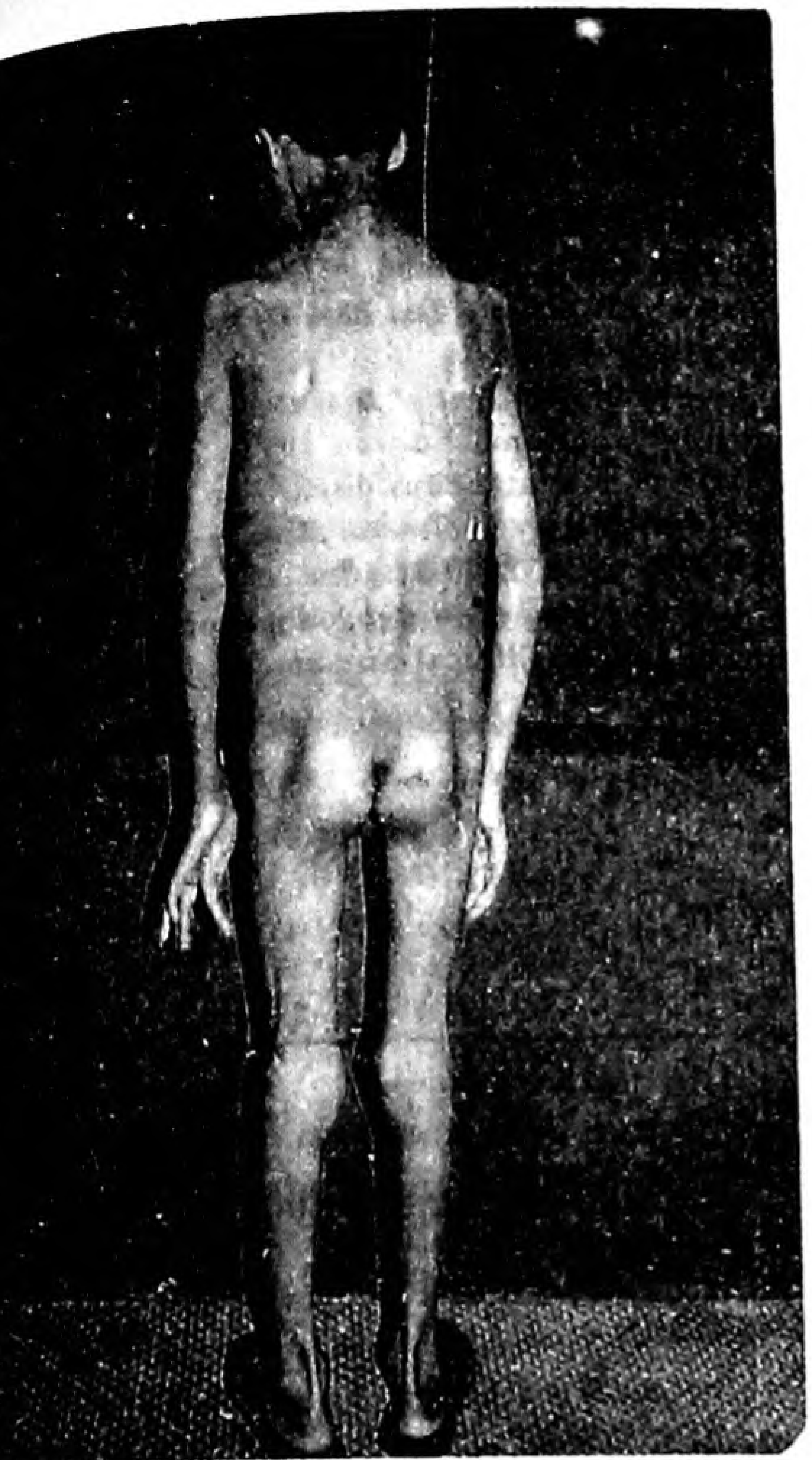

Fig.1. Multiple swellings and *cafe-eu-lait* spot

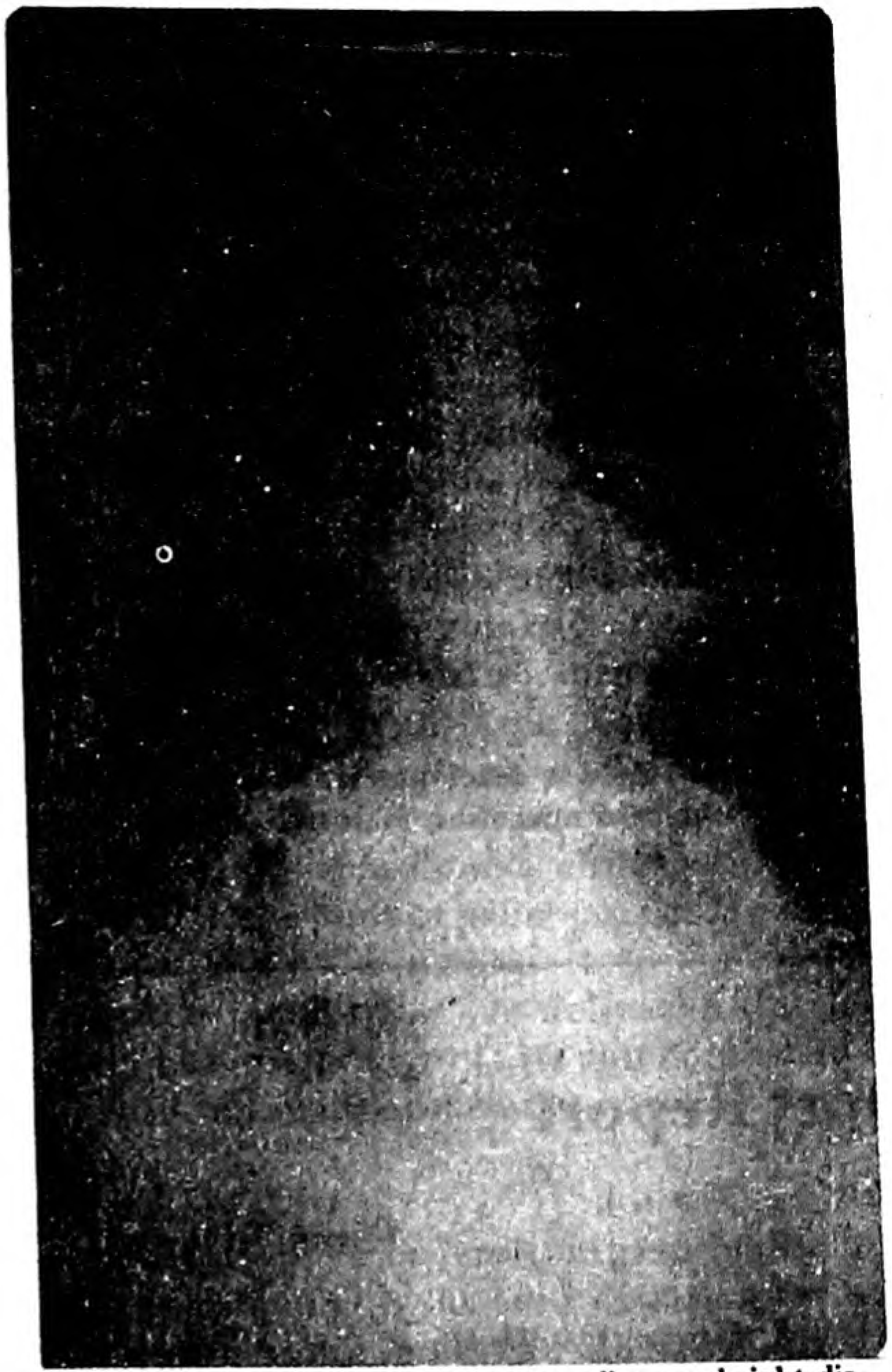

Fig.2.Interposed colonic gas between liver and right diaphragm *(Chilaiditis sign)*

the presence of Lisch nodules on iris. Auditory acquity was normal.

The hemoglobin was 8gms/dl, ESR 60mm at 1 hour, total leucocyte count, and platelet count were normal with predominant lymphocytosis. Peripheral blood film was hypochromic with micro and macrocytes. Urine examination revealed normal. Radiograph of chest and abdomen showed a colonic gas shadow interposed between liver and right diaphragm. (Chilaiditi's sign - Figure-2) Skeletal survey did not show any osseous lesion. B.C.G reaction was 12mm induration within 48 hour. Ascitic tap revealed a straw colored that lymphocytic exudate. Biopsy from one of the nodules confirmed neurofibroma. C.T Scan of head was normal. The child was started on four drug regimen for his abdominal tuberculosis and is on our regular follow up.

Discussion

Neurofibromatosis is an autosomal dominant disease. In the present case no family member was affected, therapy representing a possible new mutation [1]. The patient had neurofibromas and multiple cafe-au-lait spots. Out of various known skeletal association like congenital bowing, pseudoarthrosis of tibia, cysts of long bones, overgrowth of bone and soft tissue, scoliosis, megaencephaly, malformation of greater wing of sphenoid (1,2,5), this boy had bowing and pseudoarthrosis of left tibia, and 1% of patients with neurofibromatosis have this feature [2].

Chilaiditis syndrome, i.e. an interposition of colon and the hepatic flexure between liver and right diaphragm. It is common especially in mentally retarded or psychotic patients with chronic colonic enlargement [4,6].

Our patient was diagnosed as a case of von-Recklinghausen disease with Chilaiditis syndrome and abdominal tuberculosis. On review of literature a combination of neurofibromatosis with Chilaiditis syndrome is not reported. The two entities have nothing in common as regards to etiology, presentation or else. However, the two associated in the same patient has never been described. It is therefore of interest to have them together in the same patient.

This child was put on antitubercular treatment, given his unimmunized status, strongly positive B.C.G tuberculin test, positive family history and suggestive ascitic tap report and is being followed up for development of complications, according to NIH Consensus Development Conference for Type 1 neurofibromatosis [1]. The latest follow up available is unremarkable.

Acknowledgement

We thank all the colleagues at Children Hospital Srinagar for their assistance.

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Short Report

JK Practitioner 1995;2:198-200.

Rare Sites of Hydatid Disease

M.S. Misgar, Gh. Hussain, Qazi Massarat, Arshad Shafi.

Hydatid Disease has a worldwide distribution. The most common site affected by the disease are liver (75 percent) and lung (15 percent). In 18% subjects it passes these barriers and settles anywhere in abdomen [1]. The rare sites other than which are mentioned above are encountered in different areas [2-11]. We present 5 cases where the hydatid cysts had lodged at unusual sites.

Case reports

Case I:

35 year old man presented with a neck swelling of 2 years duration without any history of trauma or infection. On examination there was a smooth well defined non tender 4 cm^2 swelling in left lobe of the thyroid gland. Rest of the clinical examination was normal. Laboratory investigations are normal. CT was not available.

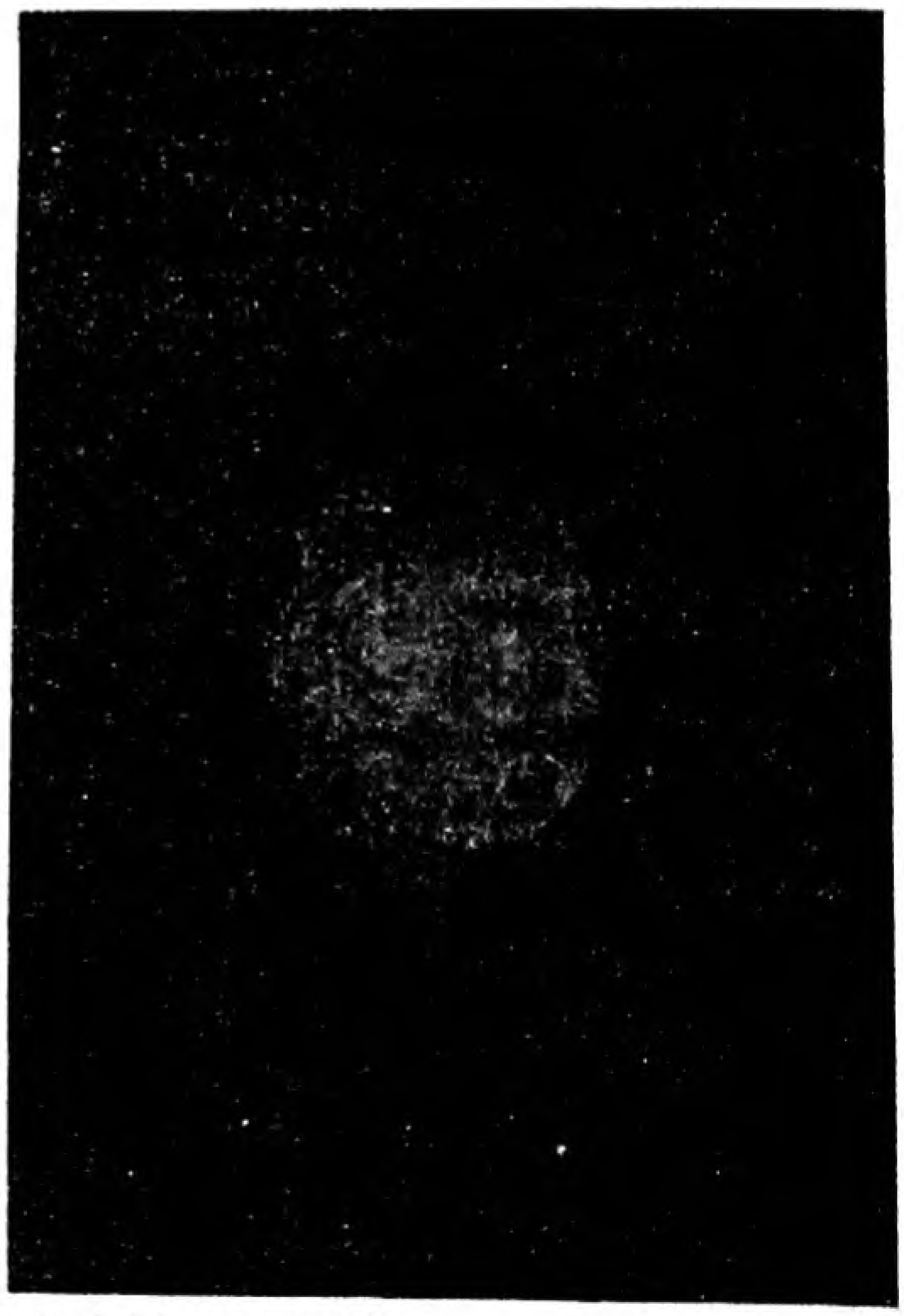

Fig.1. Hydatid cyst thyroid

On surgery exposure of thyroid gland revealed a white cystic swelling arising from lower pole of left lobe of thyroid gland. Examination revealed

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the characteristic white elastic ectocyst. On incising the wall curled and the cyst contained four small daughter cysts with clear fluid. Histopathology confirmed the diagnosis of hydatid cyst.

Case II:

A 9 year old boy was admitted with a swelling in left inguinal region of one year duration. Examination showed well defined extra-abdominal, smooth swelling measuring 3/2 inches. Laboratory investigation were within normal limits. Casoni's test was negative. Excisional biopsy was performed. A cystic swelling was excised from the lower arched portion of internal oblique muscle. Section of the cyst showed a white laminated membrane. Histopathological examination confirmed the diagnosis of the hydatid cyst.

Case III:

A 38 year old female presented with gradually progressive and intermittently painful swelling in upper part of right thigh of 6 months duration. On examination a well defined, smooth tender cystic swelling measuring 3 inches in diameter in the upper part of right thigh. Laboratory investigations were within normal limits except for an eosinophilia of 8 percent. X-ray of right femur showed multiple cystic areas in upper shaft. With the diagnosis of abscess, incision was given over the swelling and multiple cyst like structures with clear fluid came out. The cavity was found to be communicating with the medullary cavity of the upper shaft of right femur, which was curetted out. The section of the cyst showed laminated membrane. Histopathology of the specimen confirmed the diagnosis of hydatid cyst.

Case IV:

10 year old boy was admitted with history of swelling in hypogastric region of two years duration. Examination showed intra-abdominal, well defined smooth, non tender, tense, sounded swelling measuring 2 inches in diameter. Laboratory investigations were within normal limits. Patient underwent laparatomy. In exploration there was white, cystic swelling, arising from outer surface of superior surface of urinary bladder. Excisional biopsy of cystic swelling was performed. Histopathological examination of cyst confirmed the diagnosis of hydatid cyst.

Case V:

A 40 year old man was admitted with history of low back ache of six months duration and swelling in the middle of the upper abdomen of two months duration. With the appearance of swelling patient used to experience postprandial fullness.

Local examination revealed a well defined, intraabdominal non tender, firm swelling with smooth surface. 6x4" in dimensions. Laboratory tests were within normal except for an eosinophilia of 8 percent. X-ray abdomen showed a diffuse soft tissue shadow in epigastric region. Barium X-ray of stomach showed a compression of lesser curvature of the stomach with irregularity of lesser curve. Gastroscopy showed extrinsic pressure effect on posterior-surface of stomach. Casoni's test was negative. A clinical diagnosis of pseudocyst of pancreas was made and patient was subjected to laparatomy. On laparotomy a white cystic swelling arising from the body of pancreas. On incising the characteristic white ectocyst with typical white laminated membrane was delivered out completely. Histopathology of cyst wall confirmed the diagnosis of the hydatid cyst.

Discussion

The frequency of different organs in the body which are affected by hydatid disease have been variously reported Ghooi and Sikdar reported incidence of disease as under: Liver 76.8%, Lungs 9.4%, Spleen 2.1%, bones 0.9% Brain 0.7% and other sites as 2.2% [2]. In India hydatid disease is rather common [7] and several single cases of rare localization have been reported such as thyroid [4,5], bones [9], muscles and urinary bladder. Hydatid disease of urinary baldder is extremely rare. The disease is also not manifested in human before age 20 to 30 years, because of its long slow silent course. Osseous involvement is uncommon and involvement of long bones such as femur is rare. In a series of 1874 cases collected by the Hydatid Registry of Royal Australasia college of Surgeons, the incidence of bone involvement was 2.4%; pelvis is considered to be commonest bone to be affected. Bone involvement has been seen in 2%, of all hydatid disease and of these 3.4 percent were in skull bones [10].

The mechanism as to how hydatid cyst reaches distant sites like thyroid, bones, muscles and urinary bladder and pancreas is unknown and is yet a matter of investigation. Negative casoni's test is present in 5 percent of cases and eosinophilia is more associated with complication such as rupture of the cyst [11].

Hydatid cyst should be considered in the differential diagnosis of all swellings, even at remote sites.

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How to do it?

Sampling for ABG Analysis

(JK Practitioner 1995;2:201-3).

Dilafroz Bhat

Correct techniques of sampling are mandatory for accurate assessment of the parameters in the ABG analysis. Detailed below are correct techniques for the procedure.

Sampling Procedures

Blood sampling should be performed by authorized personnel only. Always handle blood samples and collection devices with cone. To avoid direct contact between operator and sample, use approved rubber gloves. Sterile technique is required at all times to avoid infection of the collection site.

It is important to approach the patient calmly and draw blood expeditiously, causing as little pain as possible. The sampling procedure should be initiated only when the patient is relaxed or the measured results may indicate hyperventilation.

Anticoagulants

A very important factor is preparing blood samples for pH and blood gas measurement is the selection of a suitable anticoagulant. The anticoagulant prevents the coagulation of the sample by defeating the clothing mechanism. Additionally, in liquid form, the anticoagulant fills the "dead space" of the syringe and in standard glass. Syringes forms a seal between the plunger and the barrel, preventing ambient air contamination of the sample.

A heparin salt is the only acceptable anticoagulant for a blood sample for pH and blood gas analysis. It is important to use the proper type and concentration of heparin; too little will allow clot formation and create subsequent instrument problems, and too much or the wrong type of heparin will cause interference with measurements. Use of an excessive volume of liquid heparin will cause dilution errors in pH and blood gas analysis. The following are the recommended heparin volumes for various samples:

Test tubes =10- 20 IU/ml blood
Syringes =30-100 IU/ml blood
Capillaries =50-250 IU/ml blood

A higher heparin concentration is recommended for syringes and capillaries than for test tubes, due to the slower mixing of blood and anticoagulant in these sample containers.

Collection site

Arterial blood or arterialized capillary blood is the preferred specimen for acid-base and blood gas studies. It is recommended to obtain blood from the radial artery, since the hand has collateral circulation via the ulnar artery, but the brachial and femoral arteries may be used, too. The neonate's temporal artery is another good site.

It is advisable to test the radial and ulnar arteries for their potency. This may be done using the Allen test; compress the ulnar and radial arteries in the wrist, while the patient clenches his fist (to drain blood) and then release the ulnar artery and watch for a flush to appear in the now opened hand. The potency of the radial artery is checked in the same way when it is released instead of the ulnar. If the ulnar or radial artery is occluded, the hand will only partially flush. In that case, another site for arterial sampling is preferable. Femoral punctures involve for more risks than other sites and should therefore be avoided whenever possible.

Venous blood is affected by peripheral circulatory efficiency and cellular metabolic needs, but may be used to reflect the patients acid-base status (pH, PCO_2 and HCO_3). A convenient puncture site for venous blood collection is the brachial vein. Capillary blood may be obtained from the digit, heel, toe or earlobe.

Collection Devices

Needle A 20-25 gauge needle or an indwelling needle [courmad type] may be used. The needle must be very sharp with a smooth beVel. A short needle for radial punctures is generally easier to handle to avoid perforation of the posterior wall of the artery.

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Syringe A sterile glass syringe having a matched barrel and plunger is ideal. A glass syringe has a distinct advantage over normal plastic syringes in that the pressure of the arterial flow will cause the plunger to withdraw on its own, indicating an arterial sample. The color of the sample is not always a good indicator of arterial blood. Patients with chronic lung disease and decreased pO_2 values will have darker arterial blood than normal.

Plastic syringes tend to have very tight fitting plungers and care must be taken to avoid creating a large negative pressure on the blood sample when the plunger is pulled back. Sampling syringes for anaerobic collection of the sample are commercially available.

Capillary tube Capillary tubes may be used for patients from whom it may be difficult to obtain arterial samples, such as newborns and geriatric patients, but only if peripheral perfusion is not impaired. The sample must be free flowing from an arterialized site. The results should then compare well with arterial samples. pH ± 0.010 and pO_2 ± 1.5 mmHg. PO_2 values below 100mmHg seem to correlate well, but above that value they become inaccurate. Patients receiving ventilation assistance [Supplemental O_2], with peripheral vasoconstriction, or in shock should not be monitored via capillary samples.

Storage and stability

All samples for pH and blood gas analysis should be tested as soon as possible after sampling. If this is impossible, the sample should be stored in ice water (2-4°C) to slow down cell metabolism. Ice cubes should not be used alone, as these may freeze and hemolyze the blood cells. Correctly iced samples that are stored upto 3.5 hours, will give reliable results. Samples with expected pO_2 values greater then that of ambient atmospheric air should, however, be analyzed immediately.

Interfering substances

No substance in normal blood will interfere with pH and pcO_2 measurements. Blood from patients anesthetized with nitrous oxide or halothane may give unreliable pO_2 values due to the influence of the anesthetic gases on the pO_2 electrodes.

Patients who have received lipid therapy will have an abnormally high blood lipid content, which causes some interference in pH measurements. Samples from such patients should be labeled so that these contaminants can be taken into account in the interpretation of the results.

Special sampling precautions

1.The area for capillary sampling must be hyperemic when the puncture is performed and during sample collection. Failure to attain proper hyperemia will result in an admixture of venous and arterial blood. Po_2 results especially at high pO_2 values will be erroneous (too low) pH and pcO_2 will not be greatly influenced.

2.Milking the area of a capillary puncture will result in an admixture of blood and tissue fluid resulting in lower pH values and higher pcO_2 with lower pO_2 and O_2 saturation. Milking also causes hemolysis.

3.If the blood is allowed to run along the skin surface, it will be exposed to ambient air. The pcO_2 will fall and the pO_2 will vary depending on the pO_2 of the patients blood. It is therefore important to use a thin smear of water soluble sterile ointment; like K-Y jelly, which causes the blood to form a nice bead.

4.Air bubbles should be removed from the sample immediately. An anaerobic sample will give the most reliable reflection of the patients condition.

5.Plastic syringes which allow diffusion through their walls once unreliable.

6.Inadequate mixing of the sample with anticoagulant is perhaps the most commonly encountered problem in the pH/blood gas field. Mixing should be thoroughly.

7.Compression of the puncture site must be maintained for at least 5 minutes. Failure to guard carefully against hemorrhage or formation of a thrombosis may result in the loss of a hand, arm or leg.

8.Usage of a local anesthetic in the sampling area should preferably be avoided. If the patient feels pain, this is usually caused by the needle penetrating the skin and then the arterial wall. The anesthetic anesthetizes only the skin penetration pain and it may make palpation of the artery more difficult.

The commonest method of sampling and the sampling procedure is summarized below:

1.***Requirements:***Syringe (dead space 2-6%)Anticoagulant, Li or Na heparin (500 IU/ml for whole blood or plasma samples)Needle (20-25 gauge, 2 units)Tourniquet (for venous samples)Skin antiseptic e.g. 70% alcoholSterile gauze, Sponge or cottonRubber stopper or syringe end cap (stainless steel)Adhesive tape and pressure dressing.

2.Mount a needle on the syringe and draw heparin into it.

3.Change needle and flush the barrel walls, thus expel air and heparin excess through the needle.

4.Extend the patients arm. If venipuncture is to be performed, apply the tourniquet.

5.Locate the blood vessel with a finger tip;.and clean the skin area with antiseptic and a sterile gauze sponge or cotton.

6. Insert the needle in the vessel with the bevel upward. Collect blood by gently with drawing the syringe plunger.

7.Release tourniquet (if used). Remove the needle and immediately apply dry gauze or cotton over the puncture site. Maintain pressure on an arterial puncture for at least 5 minutes.

8.Apply pressure dressing over the puncture site and elevate the site for at least 2-3 minutes, if possible.

9.Expel any air bubbles from the syringe and mount a rubber stopper on the needle tip (or dismount the needle and place on end cap over the syringe hub).

10.Mix the contents by inverting the syringe or rolling it between your fingers (approx. 20 times).

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Practitioner's column

JK Practitioner 1995; 1: 204-9.

Acquired Immunodeficiency Syndrome (AIDS) - (Part-I of two parts)

GH Hajini

Definition

AIDS was defined (in 1987 by CDC-USA) as a disability of life threatening illness caused by HIV and the latest definition now (1990) defines any one with fewer than 200 lymphocytes (CD4) as having full blown AIDS. This defined requires diagnostic resources which are limited in developing countries. In such areas WHO has given the following definition of AIDS for adults.

WHO clinical case definition in adults:

(Where diagnostic resources are limited)

Existence of at least 2 major signs associated with at least one minor sign in absence of known causes of immunosuppression such as cancer, severe malnutrition or other recognized etiologies. However the presence of generalized Kaposis sarcoma or Cryptococcal meningitis is sufficient by itself for the diagnosis of AIDS.

Major Signs

1) Weight loss more than 10% of body weight

2) Ch. diarrhea for more than one month.

3) Prolonged fever for more than one month (intermittent or constant)

Minor Signs: Persistent cough for more than one month.

Generalized pruritic dermatitis

Recurrent Zoster

Ch. progressive and disseminated herpes simplex.

Generalized lymphadenopathy.

Historical Perspective

AIDS has ceased to be a mere health problem, having acquired a dimension with perhaps few parallels in human history as it affects all of us directly or indirectly. After the first reported case in USA in 1981, 10-12 million adults and a million children got infected in a matter of a decade all over the world, of which 1.7 million progressed to AIDS with vast majority of them most likely dead. About 5000 persons get infected on an average every day the world over and thus by the end, 2000AD when world was supposed to celebrate the achievement of "Health for all", it will have 30-40 million men, women and children infected with HIV with nearly 1/4th of them in Asia. Globally there may be one million adults cases and deaths every year.

In India after the first case report from Madras in 1986, about one million people were infected by Oct. 1993 as against 2-3 million expected by the end of 1996. A serosurvey carried on 19 lac samples from high risk people revealed 6.98 per thousand positivity. Increasing trend is indicated by its prevalence from 2-5/1000 in 1985-87 to 11.2/1000 in 1992 and a total number of 494 AIDS patients by Oct. 1993 to 1017 upto Dec. 1994.

AIDS Virus

AIDS caused by a virus, which was first isolated in 1983 in Paris and called lymphadenopathy associated virus (LAV); isolated in USA in 1984 and named as Human T lymphocytic virus III (HTLV-III); In 1985 international Committee of Scientists named it as Human Immunodeficiency Virus (HIV-I). In 1986 HIV-II was recognized in patients from West Africa. HIV is a member of lentvirus group of Retroviridae, which is characterized by a lifelong persistence in their host and a long period of latency before causing clinical manifestations. Other retroviruses caused disease in animals which share immunological and pathological characteristics with AIDS in human beings but are genetically and phenotypically strikingly different from HIV. HIV virion is 1000 A in diameter and has an outer membrane or envelop which is 0.014 um thick and because of its glycoprotein content (gp120 and gp41) it is extremely susceptible to destruction by heat, detergents, bleach and alcohol. Inside this membrane are core proteins and genes (designated as *gag* gene, *env* gene, the *pol* gene and *tat* gene). The internal core proteins (p18 and p24) are encoded by *gag* gene, the envelope of virus is encoded by *env* gene, *pol* gene being responsible for the production of an impor-

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Professor & Head, Department of Dermatology & Venereal Diseases, Govt. Medical College, Srinagar, Kashmir.

Correspondence to: Dr. G. H. Hajini, Professor & Head, Department of Dermatology & Venereal Diseases, Govt. Medical College, Srinagar (Kashmir)-190 009. *Accepted July 31, 1995.*

tant enzyme-reverse transcriptase, which enables the virus to manufacture DNA from RNA. The *tat* gene appears to play a critical role in regulation of viral replication and possibly in the pathogenesis of AIDS. After entry into the cell the HIV is uncoated and during viral replication structural proteins are produced, two of which with antibodies made against them are used extensively for diagnosing HIV-I infection. These are a core protein P24 and envelop glycoprotein Gp-41.

HIV-2: is a virus related to but distinct from and less pathogenic than HIV-I and was first identified in 1985 from west Africa where it is endemic, and is isolated from asymptomatic individuals, AIDS patients, AIDS related complex (ARC). Core protein P24 cross reacts with that of HIV-I but envelop glycoprotein gp-36 of HIV-2 is type specific and enables distinction between the two infections. Thus individuals infected with HIV-2 often do not give a positive reaction to enzyme immune assay used for HIV-I infection as antibodies against envelop glycoprotein gp 41 of HIV-I are usually not produced while most individuals infected with HIV-2 have antibodies that cross react with corporation of HIV-I in Western blot assay. However currently used serological tests increasingly incorporate antigens capable of eliciting antibodies to both HIV-I and HIV-2. HIV-2 also infects preferentially CD4+cells and follows the same mechanism of transmission as HIV-I.

HIV infection of CD4+cells may result in cell destruction, or the virus may also remain in a state of latency in lymphocytes or macrophages or replicate without causing clinical disease. The cytopathic effect on infected cells is not known. But once an individual has become infected with virus, he remains infected for life.

HIV has been isolated from blood (serum, CD4+cells, plasma) semen, vaginal/cervical secretions, bone marrow, saliva, brain tissue, CSF, urine, ammniotic fluid, breast milk but only blood, sexual secretions and breast milk have been shown to transmit the virus.

Incubation period:

I.P.between the acquisition of HIV infection and onset of AIDS is around 8-11 years. Proportion of HIV infected patients who eventually develop AIDS is not yet known but in one study from USA 54% had AIDS within 10 years of seroconversion and about half of the recipients of HIV contaminated blood developed AIDS within 7 years.

Transmission:

Risk factors include multiple heterosexual partners, blood and blood products transfusion, use of unsterilized needles and syringes, mother to fetus/infant transmission before, during or shortly after birth, STD's in particular ulcerative genital lesions, male homosexuality, and IV drug abuse. Hemophiliacs are comparatively much less important factors in Africa than in USA, Europe and Australia. Systemic factors that may influence infectivity during sexual contact is the clinical stage of HIV infection since the transmission rate to unaffected partners seems to be highest for patients with more advanced clinical disease and/or lower levels of CD4+cells.

HIV survives poorly outside human body and is transmitted mainly via blood and semen. Thus the main routes of infection are a) sexual b) administration of blood or blood products c) infection with blood contaminated equipments d) Maternofoetal. A person once infected remains infected and infectious for life but infectivity is greatest at the time of initial acute illness and later during clinical illness with low levels of T helper lymphocytes and p24 antigenemia. In adults sexual contact is by far the dominant mode of infection followed by blood transfusion, exposure to needles and syringes.

Sexual transmission:

The infectivity is low in uncomplicated vaginal intercourse compared to other STD's; the risk of contacting the virus in any such encounter with an infected partner has been estimated at 1: 500. Mucosal trauma (more likely in anal intercourse) and other ulcerative diseases of genitalia facilitate viral infection. Orogenital infection is difficult to prove, probably possible, but less important than anal and vaginal routes. Transmission through artificial insemination has occurred. In children the dominant mode of transmission is perinatal with secondary role for blood transmission.

Transmission through blood and blood products:

Most patients transfused with blood later found to contain HIV, became infected. Avoiding donors from high risk groups and prior HIV antibody testing of donated blood has greatly reduced but not totally eliminated the risk as blood donated after infection but before seroconversion would pass undetected. Over 50% hemophiliacs in a large American study became infected with HIV before the virus and its mode of transmission were discovered. Heat inactivation of factor VIII concentrate appears to be completely effective.

IV drug abuse: Sharing of injection equipment has lead to higher prevalence of HIV infection among drug abusers.

Health care workers:

Occupational HIV infection is rare. Main risk is in occupation of blood during needle pricks or similar injuries, the risk of infection per exposure is estimated at about 0.5%.

Maternal Transmission:

The exact frequency of perinatal transmission from an HIV infected mother is unknown. In Africa the rate varies from 20-60% among children born of HIV seropositive mothers. In areas where women between 20 and 29 years of age have high prevalence rates, an important proportion of children may already be infected with the virus at birth or acquire infection during or shortly after birth. Infection usually occurs transplacentally or though unproven, at delivery.

Breast feeding:

Reports of occasional cases of apparent transmission through breast milk are there. Mostly these involved mothers infected by post natal blood transfusion, so that infection probably occurred from women viraemic, but as yet without antibody to babies without passively acquired antibody. Transmission in breast milk is less easy to prove from women with preexisting HIV infection and antibody to their antibody bearing baby but risk is low compared with that of per or intrapartum transmission in such cases. Since breast milk is the best baby food in the world giving nourishment and protection from many communicable diseases and since HIV is known to be secreted in breast milk and can cause infection to unaffected by HIV positive mother, WHO recommends that in areas where primary cause of infant deaths are infectious diseases and malnutrition, breast feeding should be a standard advice to pregnant mothers including those known to be HIV positive as the risk of infection through breast milk is likely to be lower than the risk of dying of other causes if deprived of breast feeding; but in areas where infectious diseases and malnutrition are not significant causes of infant mortality safe feeding alternative should be adopted.

No evidence exists to support any other route of including through food, water, vectors or a casual contact. HIV has been identified in whole blood, serum,tears,saliva, CSF, brain tissue, urine,semen,breast milk, ammniotic fluid,vaginal/cervical secretions, but low title of virus and infrequency of virus isolation from most of these fluids suggests that except blood,semen,cervical/vaginal secretion and breast milk they play an insignificant role in HIV transmission. breast milk they play an insignificant role in HIV transmission.

AIDS and Women

At the start of AIDS the victims were mostly homosexual men. In Africa and S.E.Asia heterosexual rather than homosexual pattern altered the perception. WHO has estimated that by the end 2000,HIV infected women will be 13 million and 4 million will be dead because of AIDS. In India is mostly through heterosexual contacts so coming years will see more women infected in reproductive age group. It is known that 2/3 children born of seropositive mothers escape infection. These children run the risk of death of one or both parents in next 10 years. Thus women and children will move from periphery to the center of concern. Women compared to men are more vulnerable to infection for reasons:-

a)***Biological:*** All body fluids in infected person contain virus but with different concentrations and chances of infection depend partly upon this concentration. There is a higher risk of women contacting infection from seropositive male partners as compared to unaffected males from seropositive women. Besides as a receiving partner she a large mucosal surface exposed during sexual contact.

b)***Epidemiologic:*** Women marry generally older men, who have generally more premarital sexual experience which puts a woman at a higher risk of getting infection from their spouses. Besides child bearing may necessitate medical intervention including blood transfusion which again increases the vulnerability.

c)***Social reason:*** Most of the women are economically dependent on men. Economic support automatically entitles a man to have sexual privilege. Even on the use of condom by a female, final say lies with the male. This reflects the poor social status of women in society.

HIV does not spread by:

1.Drinking water from same glass as used by an infected person.2.Swimming in pools used by people with HIV or AIDS. 3.Getting bitten by a mosquito that has already bitten an infected person.4.Getting bitten by infected person. 5.Casual living with people with HIV or AIDS.6.Caring and looking after people with HIV or AIDS.7.Use of same toilets as AIDS patients or people infected with HIV.8.Shaking hands with people with AID or HIV infection 9.Hugging or kissing a person with HIV or AIDS 10.Casual contact as sitting together, coughing, sneezing, or from water, food, clothing, chips glasses, plates, forks,spoon and other shared objects. 11.Donating blood12.Bed bugs, flies lice, please, other insects and pest do not spread HIV.

HIV infection and pregnancy

Pregnancy in a seropositive woman does not alter the natural history of the disease. There is no increased risk of developing clinical AIDS in a seropositive woman. Pregnancy does not accelerate the eventual death in clinical AIDS. Chances of fetal infection depend upon the seropositive status of the women. In vitro infection of fetus is high if mother has AIDS. It is also high if mother contacts infection while she is pregnant. Risk is also higher if mother delivered HIV positive baby in her earlier pregnancy. Chances of in vitro infection of fetus is low if mother is already seropositive put does not have AIDS when she conceived the child.

Prevalence

In central and East Africa AIDS is equal in males and females in contrast to 19:1 ratio in USA. Age wise AIDS incidence and seroprevalence is at its peak between 20-39 years of age (women generally being younger than men) AIDS is predominantly an urban problem in most countries, however it is high in rural areas in some Africa, countries.

HIV-2 infection is fairly well confined to west Africa where seroprevalence in general population ranges from 0.3-17% (contrast to 1-20% for HIV-1 seroprevalence in other African states in urban areas). HIV-2 infection is transmitted through same routes as HIV-I and infects same population with sexual transmission among heterosexuals being the dominant mode spread. Upto 64% of individuals at risk for STD such female prostitutes were found to be HIV-2 seropositive in one study. HIV-2 can cause disease in humans as indicated by its isolation from blood and CSF from patients with AIDS and ARC.

Pathogenesis

Progressive immune dysfunction marked by deficiency of cell mediated and humoral immune responses and loss of immune surveillance is the hallmark of HIV infections. Microbes that are present in all persons but unable to cause disease can proliferate unchecked resulting in opportunistic infections. Transformed cells which are detected and destroyed by a healthy immune system remain undetected and proliferate causing malignancies. Indeed many infections and cancers seen in AIDS patients were previously known only in patients born with defects of the immune system, had been impaired by cancer chemotherapy or use of immunosuppressive drugs for organ transplantation e.g. pneumonia due to infection by *Pneumocystis carinii*, and Kaposi sarcoma. How can a virus, smaller than the size of the smallest bacterium destroy the whole immune system and lead to death. The answer lies in the intelligent strategy of the HIV paralysing the immune system.

The entire spectrum of immune deficiency seen in HIV infected patients can be traced back to a single cause i.e.loss of T4(CD4) helper lymphocyte function. The normal immune response depends on a coordinated interaction between antigen presenting cells, T-cells and B-cells. The antigen presenting cell include macrophages and dendritic cell e.g. Langerhans cells in the skin and interdigitating cells in the lymph node. These cells engulf and degrade antigen and present it to a subset of T lymphocytes called T4 (CD4) helper cells. These cells are so named because they have a specific surface marker (antigen) on their cell membrane which is bound by monoclonal antibodies. This marker is not present on other T lymphocytes. They instead have a CD8 marker and called T8 cells. The T4 cells after receiving information about the antigen from the antigen presenting cells provide help to the T8 cells which are responsible for cells mediated immunity and to B cells which secrete immunoglobulins and are responsible for humoral immunity. The immune response occurs most effectively in structured lymphoid tissue e.g. in lymph nodes and spleen as well as unencapsulated but somewhat structured areas like lymphoid tissue in tonsils, Peyers patches and appendix referred to as MALT (mucosa associated lymphoid tissue). Dysfunction of T4 helper cells thus impairs both cell mediated and humeral immunity.

The HIV recognizes the CD4 molecules present on the cell membrane of the T4 cell and binds it. The gp 120 protein in the envelope binds to CD4 protein on T4 lymphocyte membrane. The viral envelope membrane fuses with the cell membrane of the lymphocyte and the vital core is released in the cytosol. This is followed by multiplication of the virus and destruction of the cell. This results in a drastic fall in peripheral blood T4 count. In normal persons T4 cells make up 60-80% of the circulating T-cells population. In AIDS they may become undetectable. As T8 cells are unaffected the T4/T8 ratio also falls markedly. Apart from depletion of T4 cells the HIV somehow causes loss of function of surviving T4 cells so that immune system may become weak even though the number of T4 cells may still be normal. Lacking help from T4 cells the B cells cannot produce adequate antibody to specific antigens. Macrophages and natural killer cells cannot function properly. The T8 cells needed for cell mediated immunity also can not function and thus both humeral and cell mediated immunity is greatly impaired. The depletion of T4 cells also disrupts the structured lymphoid tissue of lymp nodes. Fusion of infected T4 cells with normal cells spread virus to other tissue because virus promotes fusion of infected cells with other cells. Infection of cells of mononuclear phagocytic system (monocytes, macrophages, glial cells etc.) by HIV can also impair the function of these cells. Infection of dendritic cells in follicles of lymph nodes may alter then to cause T4 and antigen independent stimulation of B-cells. This is called polyclonal activation of B-cells and causes the B cells to secrete large amounts of antibody. This can cause hypergammoglobulinemia. However as the secreted antibody is non specific i.e. it is not directed against a particular antigen it can not help in providing immunity. A subset of T8 lymphocytes called T suppressor cells are needed to taper off the immune response. Without help from T4 cells the suppressor cells can not function so that B cells continue to secrete non specific antibody. Finally the ability of HIV to infect the central nervous system may be responsible for psychoses and brain atrophy seen in AIDS patient. During prolonged asymptomatic phase of infection, the immune system keeps these effects of virus in check, though there is gradual decline in T helper cell count from the normal of about 800/mm^3. The events that finally cause the defense to fail are not known but when it does the symptomatic phase of HIV infection begins. With the decline of immune function there is increasing incidence of skin changes so that ultimately nearly all patients will have significant dermatologic disease.

Nature history of HIV-I infection

Contact with HIV
Infected Not infected
asymptomatic carrier
AIDS
persistent generalized lymphadenopathy
Non-life threatening HIV Disease.
ARC Death.

Soon after becoming infected with HIV-I some people have an acute self limiting illness indistinguishable from many other mild viral illnesses. After a healthy carrier state which may last as long as 10 years most infected people develop long term clinical illness due to HIV-I or opportunistic agents. Among seropositive HIV-I infections 2-8% per year develop AIDS. Thirteen years after infection about 56% will have developed AIDS. In Europe and America cases fatality rate of AIDS approaches 100% within 4-5 years after the diagnosis.

With HIV-2 infection incubation period seems to be longer and rate of progression towards ARC and AIDS slower.

HIV infection has 4 phases:

Phase I:

Acute retroviral infection: This phase is usually asymptomatic and can pass unnoticed but can behave like acute mononucleosis like illness in about 50% infected people, characterized by lethargy, malaise, fever, sore throat, myalgia, anorexia, sweating, arthralgia, headaches, diarrhea, nausea, generalized lymphadenopathy,macular eruption on trunk and arms, thrombocytopenia, occasional transitory aseptic meningoencephalitis, mono or polyneuritis. Acute illness generally occurs 10-30 days after exposure and resolves in 3-21 days. Transient lymphocytosis with atypical lymphocytes may be seen. Antibodies had not yet appeared but P24 antigens can be detected for 1-2 weeks.

Phase II:

Asymptomatic HIV infection: This latent period may last for upto 10 years after acquisition of infection. During this phase patient is probably infected since HIV can be isolated from peripheral blood lymphocytes. IgG antibodies to HIV can be demonstrated in serum and in some cases in CSF. Immunological tests show that the numbers of lymphocyte sub-population and their functions are normal. Towards the end of this phase neurological dysfunction may be detected.

Phase III:
Persistent generalized lymphadenopathy: other than P.G.L. there are no other signs and symptoms of infection. There should be lymph nodes enlargement in 2 or more extrainguinal sites for at least 3 months. Nodes are rubbery and mobile and may be intermittently tender. Antibodies to both P24 and GP 41 are detectable.

Phase IV:
Symptomatic HIV infection: At the beginning of the phase there may be a decline and disappearance of antibody to P24,followed by the reappearance of P24 antigen in blood, antibodies to gp41 continues to be produced. Majority of the patients have decreased number of T helper cells usually below 100/mm^3.' Progressive impairment of immune function will lead to full syndrome of AIDS. AIDS is thus defined as occurrence of major disease indicative of a defect in cell mediated immunity in patient with no known cause for immunodeficiency other than HIV infection. Great majority of such patients are seropositive for HIV but a few are not, thus seropositivety is not essential for diagnosis. Most common clinical manifestations are weight loss and weakness. Weight loss is often the first sign and in some patients maybethe only sinuously. Temporary weight gain with nonspecific treatment is not long lasting. Accelerated with the loss in HIV infected patients in a sign of disease progression. Some AIDS patients loose over 40% of their weight and most die in extreme cachexia. In most patients no specific cause can be identified for weight loss. About 50% of AIDS patients have episodic fever lasting more than a month, which is usually low grade and intermittent, with chills and night sweats and thus may be caused by infection, malignancy, drug therapy or unknown causes. Thus the signs and symptoms can be summarized from a African study as:

1.Weight loss of more than 10% of body weight.
2.Fever of more than one month.
3.Diarrhea of more than a month
4.Headache of more than a month
5.Weakness
6.Cough
7.Pruritis
8.Dysphagia
9.Dyspnea
10.Anorexia
11.Thrush
12.Popular prurigo
13.Generalized prurigo
14.History of zoster
15.Neurological abnormalities
16.Focal neurological disorder.

ADVERTISEMENTS ARE ACCEPTED ANY TIME, BUT SHALL BE PUBLISHED ACCORDING TO THE AVAILABILITY OF SPACE IN FORTHCOMING ISSUES.

Medical Curricula in India

(JK Practitioner 1995;2:210-11)

Learning Anatomy in Primary Care Curriculum

Dr. M. Aslam,

The learning process of present day medical students in all the subjects including Anatomy, is exam oriented rather than putting in this knowledge thus acquired in their actual practice of the medical profession.

Time:Though the time period allotted for learning of anatomy was reduced from 2 years to 1 1/2 year; yet no modifications have been made in the syllabi thus throwing the medical students in wilderness. The matter became still more complicated when we go in details of actual teaching hours during this period of 1 1/2 year. The prescribed allotted time as laid down in by M.C.I is gravely affected in my state as will be revealed as under. I blame the bureaucrats and the politicians of the times for their vested interests.
Declaration of list of admission in November of given year.

-Actual admission in College and couple of classes-for 2 weeks of December of year A
-Winter vacation from 15th December of year A to Feb of year B
-Summer vacation in year B
-Winter vacation in year B
-Sundays in A & B
-State/Central gazetted holidays
-Political disturbances etc.
-Terminal exam.

Thus it is amply clear that neither the teacher nor the students can do justice to their respective jobs of teaching and learning.

Books, curriculum & methodology of teaching
It is unfortunate that there is no apparent change in the curriculum ever since the Britishers left and we inherited their system though our needs and demands may have been different. This is evident from the fact that we still use the Cunningham's dissectors and no alternative dissectors have been made available in India which boasts of over 130 medical colleges, while I must admit that some of the Indian authors have written excellent text books which are very popular especially with the students. Unfortunate part of this learning process in India is that every teacher teaches anatomy as a morphological science, thinking he is making an anatomist and not a basic doctor and as such ignores that he should impart teaching of anatomy as a functional unit. Since learning process in our country is more exam oriented with the result, by the time the student goes to higher classes, he forgets the fundamentals of anatomy. Teachers by and large go to finer details with the result in this age of every day to day increase in the knowledge of medical sciences the student becomes victim. In my opinion, the dissection's hours especially in H & Neck may be curtailed this being purely a specialist field of a dentist, ENT and some amount of allotted time for head & neck could directly be demonstration protected parts, lecture demonstration, audio-visual cassettes and clinical demonstration by otolaryngologist, ophthalomologist and the dentist. Thus enabling the doctor in a remote village to examine, handle, treat and in other case direct the patient to the correct quarters. Likewise the histology should be restricted to the extent that students should be able to identify the tissues of general and special system of body with their salient features rather than long dialectical lectures prepared from Ham and Fawcet, Bloom, as though we are preparing a histologist. Radiographic/Living Anatomy is under-rated in present course and marks allotted are much less in an examination. There are a varied range of topics/regions/systems where the anatomist could sit, discuss and restructure the syllabi thoroughly and conclusion could be arrived at.

Evaluation of learning
Process is through written and oral examination. It is unfortunate that no uniform pattern is being adopted in whole of country. While the northern region, still holds to old traditions of exams while is southern states, the semester system has been adopted by and large. Thus the students from northern region has to learn and prepare for whole course while his southern counterpart has to learn and qualify the course by piece-meal, thus

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J.V.C.M. Sciences Srinagar.
Correspondence to: Dr. M. Aslam, Jehlum Valley College of Medical Sciences, Srinagar. *Accepted: March 31, 1995.*

giving him an edge over the former though I cannot give any statistical end results of two system.

Some suggestions

1.For medical education New Education Policy to be declared by the Central/State Government prior to which seminars/discussion to be held representing members of various states university (not confined to Delhi) so that ultimately a consensus is obtained with regard to requirements as a direct study of the problem rather through M. C. I whose role now-a-day, in my opinion is ineffective.

2.Selection of the candidates at the school level immediately after they qualify the school examination on the basis of merit by competitive examination for open merit and various genuine categories, the candidate should be selected. Their course for P. U. C and 12th should be amalgamated to precise and to the point and only of one year duration. Syllabus being recast in physics, chemistry and biology with a purpose that these courses help them and are oriented to medical sciences only.

3.For doctors who are working in villages, a specific refresher course of short duration comprising of few weeks in basic sciences should be held supplemented by demonstration of cases in clinical sides. We have to think, ponder and act now lest Health for all by 2000 A.D. remains only a dream.

Change in address of
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New address: P.O.BOX 884
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MODERN ENDOSCOPY CENTER, JHELUM MARKET, BUTMALOO, SRINAGAR

DR. G. M. MALIK
M. D MACG (USA)

Meet the Consultant from UK.

Dr.Mohammad Afzal Mir, FRCP,

University Wales College of Medicine, Cardiff

Place of Birth: Trehgam, Kupwara, Kashmir

Educational qualifications

MBBS Vikram University, Ujjain, India1962
DCH, London 1965
MRCP, UK 1972
FRCP, London 1985

Present appointment

Senior Lecturer & Consultant Physician, Department of Medicine, University of Wales College of Medicine, Heath Park Cardiff CF4 4XN

Biographical listings

The Medical Directory 1995; Marquis Who's Who in the World 1987-1995; Who's Who in the Universities 1995; Men of Achievement 1987-1995; 500 Leaders of Influence 1995; The International Directory of Distinguished Leadership 1995; The International Who is Who is of Contemporary Achievement 1995; Who's Who in Medicine.

Prizes, honours and scholarships

Young Research Workers Award (British Cardiac Society) 1976

European Travelling Fellowship (British heart Foundation) 1977

Numerous first place prizes in medical school for academic and debating achievements.

Special interests

Diabetes and metabolism (obesity), sleep apnoea, cor pulmonale, ischaemic heart disease, hypertension and medical education.

Publications

Over 100 publications including some in The New England Journal of Medicine, The Journal of Clinical Investigation, The Journal of Biological Chemistry, Annals of Internal Medicine.

Books

(1) An Aid to MRCP Short Cases; (2) An Aid to MRCP Viva; (3)Atlas of Clinical Diagnosis

Communications

Over 100 communications given to various scientific bodies in Europe, USA, Asia and Africa.

Videotapes

Made 70 Videotapes on Clinical Skills for graduates which are available in the local libraries. Also made 38 videotapes for postgraduates undertaking higher examinations. These have been acquired by a majority of the postgraduate centers in the UK and by some centers in South Africa, Middle East and Australia.

Made 3 interactive discs for Clinical Skills which helps students to learn history taking.

Curriculum

Helped to devise the previous two curricula and now involved in developing the new curriculum for the University of Wales College of Medicine.

Membership

British Cardiac Society; Medical Research Society; British Diabetic Association; American Hypertension Society; British Hyperlipidaemia Association; British Hypertension Society; Marylebone Cricket Club.

Meet the Professor from U.S.

Prediman Krishan Shah, MD., FACC

Department of Cardiology, Cedars-Sinai Medical Center, 8700 Beverly Blvd. Los Angeles, CA 90048.
Date of Birth: August 29, 1949.
Place of Birth: Srinagar

Educational Qualification

MBBS, Medical College, Srinagar (1969)
Residency, University of Wisconsin, 1972-73
Residency, Albert Einstein College of Medicine, 1974-76
Research Fellowship Cardiology, Cedars-Sinai Medical Center, 1976-1977.

Present appointment

Director Division of Cardiology and Atherosclerosis Research Center, Shapell and Webb Chair in Cardiology, Professor Medicine, UCLA, Cedars-Siani Medical Center, Los Angeles, CA.

Honors & Special Awards

1.Best outgoing graduate of Medical School.
2.Recipient of Mount Sinai Alumni Association Plaque, Golden Apple and Kaiser award for excellence in teaching.
3.Fulbright Visiting Professor to Republics of Argentina and Chile.
4.Shapell & Webb Family Chair in Clinical Cardiology.
5.UCLA Dean's Faculty Award for Excellence in Teaching.
6.Visiting Associate Professor, Harvard Medical School and Cardiovascular Biology Research Lab. at Massachusetts General Hospital.
7.Annual Honoree of the Save-A-Heart Foundation.

Special Interests

1.Pathophysiology, Radionuclide assessment, Thrombolysis, Antithrombotics in Acute Ischemic Syndromes.
2.Lipids and restenosis.
3.Chronic Heart Failure.
4.Endothelial Functions.
5.Role of macropahge metalloproteinases in plaque rupture, thrombogenicity of plaque in atherosclerosis and HDL mutant in intimal proliferation in hyperlipidemic animals.

Publications

Ninety-two publications in various International Journals including some in the Chest, JACC, Circulation, Am Heart Jr, Annals of Internal Medicine and Am Jr of Cardiology.
Authored about twenty-six reviews and invited articles on various aspects of cardiac failure, ischemic syndromes and atherosclerosis.

Books

Twenty-nine chapters in Books related to Cardiology, edited Acute Cardiac Care (1991).

Communications

About a hundred communications to various scientific bodies in USA, Europe and Asia.

Membership

-Fellow of American College of Cardiology, American College of Chest Physicians, American College of Physicians, Argentina Society of Cardiology, Chilean Society of Cardiology.
-Member American Federation for Clinical Research, Council on Critical Care, Emergency Cardia-care Committee of American College of Cardiology and many other Societies related to Cardiac-care.

Editorial services and Journal References

1. American Journal of Cardiology, 2. American Heart Journal, 3. Annals of Internal Medicine, 4. Circulation, 5. Cardiology, 6. Chest, 7.Journal of Electrocardiography, 8.Heart & Vessels and 9. The New England Journal of Medicine.
- Member of the Editorial Board of: 1. American Journal of Cardiology, 2. Journal of the American College of Cardiology.

Communications to the Editor

Dear Chief Editor,

Thank you for your letter dated June 06, 1995. I have received the Journals. Thanks for electing me as a Consultant Editor in your respectful Journal.

Dr.Abdulaziz H. Al-Saigh
Medical Director
King Fahad Hospital Madina,
KSA

Dear Chief Editor,

Many thanks for the complimentary copy of your nice Journal (JK Practitioner). I do not mind being on the advisory committee of your editorial board but I doubt if I will be able to contribute substantially for your Journal because of my multiple prior commitments.

Sincerely,

Rakesh K Tandon,
Professor & Head, Department of Gastroenterology
All India Institute of Medical Sciences, Ansari Nagar,
New Delhi-110029.

Dear Chief Editor,

I congratulate you on behalf of all the colleagues and medical fraternity of Kashmir for having come out with a quality Medical Journal that was needed for a long time. I will help you in any way I can.

K.L. Chowdary
Professor of Medicine, Jammu.

Dear Chief Editor,

I want to congratulate to you for your resolve and determination to launch a new medical Journal from Kashmir despite adverse conditions prevalent in the valley. I will support your endeavor in any way I can.

Prediman K. Shah,
Department of Cardiology,
Cedars Sinai Medical Center. 8700
BeverlyBlvd,LosAngeles,California.

Dear Kashmiri Doctors:

I was pleasantly surprised to see the first edition of The JK Practitioner Journal. This has been the "brain child" of Dr. G. M. Malik, who felt the need to upgrade the skills of physicians in practice, particularly in rural areas of Kashmir. The first issue has articles dealing with hepatitis, typhoid, smoking, vertigo etc. The 2nd issue featured among many articles, a brief case report on the delivery of a healthy child by a woman with two gun-shot wounds in the uterus, "it is He who fights life and death, and when He decides upon an affair, He says to it `Be', and it is", Q40:68. I envision a very useful role for this journal. It will provide a link amongst the large number of alumni from our alma mater - probably over 3,000 by now. It can provide encouragement to the young house staff at Srinagar, and it can highlight some of the unique issues facing health providers in Kashmir.

Faroque A Khan,
Chairman, Department of Medicine,
Nassau County Medical Center, New York, USA

Dear Chief Editor

I must congratulate you that you have got this project off the ground and, considering the difficulties, your first issue was excellent. I have been giving it some thought and I feel that in its earlier stages you ought to have less emphasis on hard research and more on readable matter which would be of interest to your readers in the subcontinent. In particular, you may consider having 2 features, one on clinical practice and the other a light feature such as overseas diaries. In the former you could have an article written by one of your experts in Kashmir and then you could have it reviewed by an outside expert so that their combined thoughts would be crystallized in the papers. For example, if you had an article written on Management of Diabetes by a local expert then one of us could review it and add a few more things, the same could apply to hypertension and many other common disease in kashmir. The overseas diaries could contain a communication by a medical student in countries and sometimes by a general practitioner. These articles would reveal a medical community practice outside the subcontinent and I am sure would be reader friendly.

Dr. M. A. Mir,
Senior Lecturer & Consultant Physician,
Department of Medicine, University
Hospital of Wales, Health Park Cardiff CF4 4XW, U.K.

Dear Chief Editor,

Thank you for your letter of June 21, 1995. I am extremly grateful for considering me as one of the members of the Advisory Committee which I accept. I shall try to contribute article in your journal at the earliest.

Dr. B. Sanyal
Director
Madhya Pradesh Cancer Chikitsa
Evam Seva Samiti Bhopal

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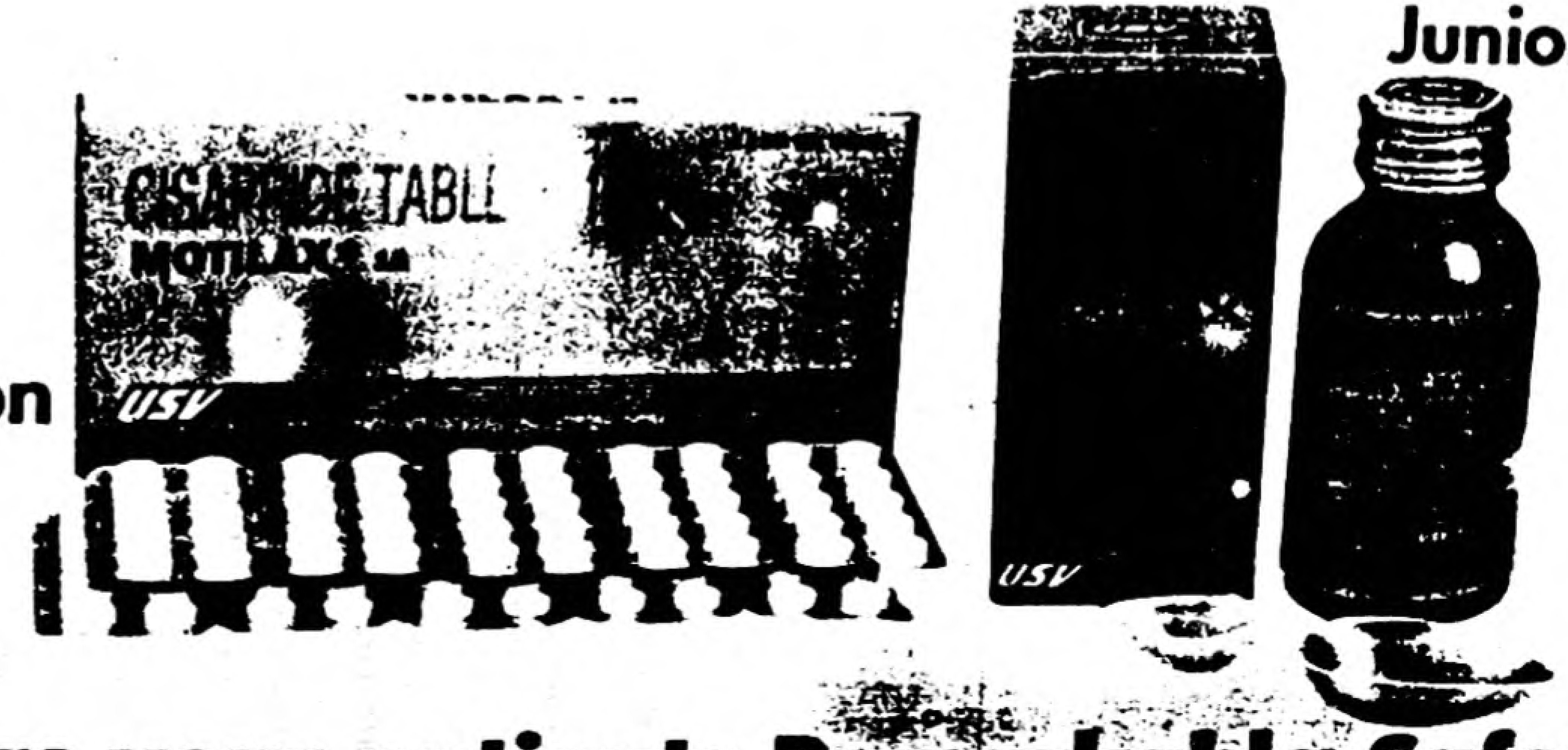

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